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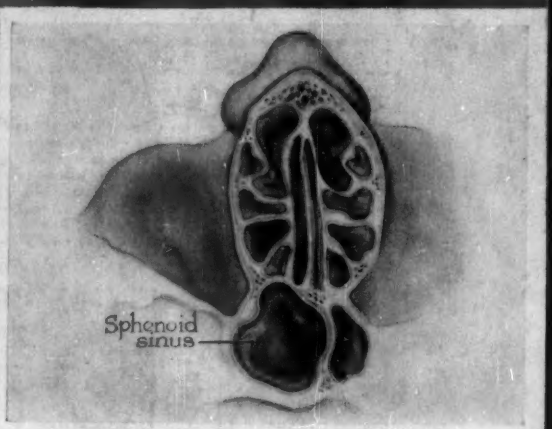

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TABLE OF CONTENTS

VOLUME 63 FEBRUARY 1956 NUMBER 2

ORIGINAL ARTICLES

PAGE

Reaction to Cocaine Used as Topical Anesthetic in Bronchoscopy 115
R. A. Tella, M.D., and Wilson Weisel, M.D., Milwaukee

Wegener's Granulomatosis
Carl W. Hoch, M.D., Cleveland 120

Gross Pathology of Sinus Discharge
Holland N. Stevenson, M.D., New Rochelle, N. Y. 124

Atresia of the External Ear Canal
Marvin W. Simmons, M.D., and Thomas J. Fuson, M.D., Fresno, Calif. 128

Use of the Electrolarynx in Speech Rehabilitation
Oscar W. Lueders, New York 133

Hypometabolism in Relation to Ear, Nose, and Throat Disorders
A. R. Hollender, M.D., Miami Beach, Fla. 135

Intracranial Complications of Chronic Mastoiditis
Harry R. Morse, M.D., Knoxville, Tenn. 142

Temporal Bone Surgery in Otitis Media and Mastoiditis
Karl Musser Houser, M.D., and Herbert J. Dietrich Jr., M.D., Philadelphia 146

The Surgical Treatment of Vertigo
Shoichi Honjo, M.D., and Ryuzo Furukawa, Ube, Japan 153

AUDIOLOGY SECTION

A New Formula for Using the Audiogram to Predict Speech Hearing Loss
J. Donald Harris, Ph.D.; Henry L. Haines, M.D., and Cecil K. Myers, B.S., New London, Conn. 158

Pathology and Audiology of Middle-Ear Lesions
H. G. Kobrak, M.D., Ph.D., Detroit 177

Speech and Voice Training of the Deaf
Esti D. Freud, Ph.D., New York 183

CASE REPORTS

Adenocarcinoma of the Parotid Duct
T. E. Beyer, M.D.; J. R. Blair, M.D., and Paul K. Hamilton Jr., M.D., Denver 196

An Unusual Case of Sphenoid Abscess
Claude C. Cody III, M.D., Houston, Texas 199

The Management of Laryngeal Keratosis by Laryngofissure
Arthur J. Cracovaner, M.D., New York, and Alan S. Rubenstein, M.D., Wilkes-Barre, Pa. 203

A Chlorpromazine (Thorazine)-Induced Cephalalgia and Its Management
Sam E. Roberts, M.D., Kansas City, Mo. 208

CLINICAL NOTES, NEW INSTRUMENTS AND TECHNIQUES

Esophageal Dilator
Harvey H. Sirota, M.D., and Vera P. Sheppard, R.N., East Hartford, Conn. 210

Otometer (A New Portable Hearing Testing Instrument)
Howard P. House, M.D., Los Angeles 211

A Bilateral Self-Retaining Nasal Speculum
Max Unger, M.D., New York 212

Bronchoscopic Biopsy Spoon
G. Zakhary, M.B., Ch.B., D.L.O., Cairo, Egypt 213

REGULAR DEPARTMENTS

Abstracts from Current Literature 215

News and Comment 157, 198, 207, 209

Directory of Otolaryngological Societies 227

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VOLUME 63 FEBRUARY 1956 NUMBER 2

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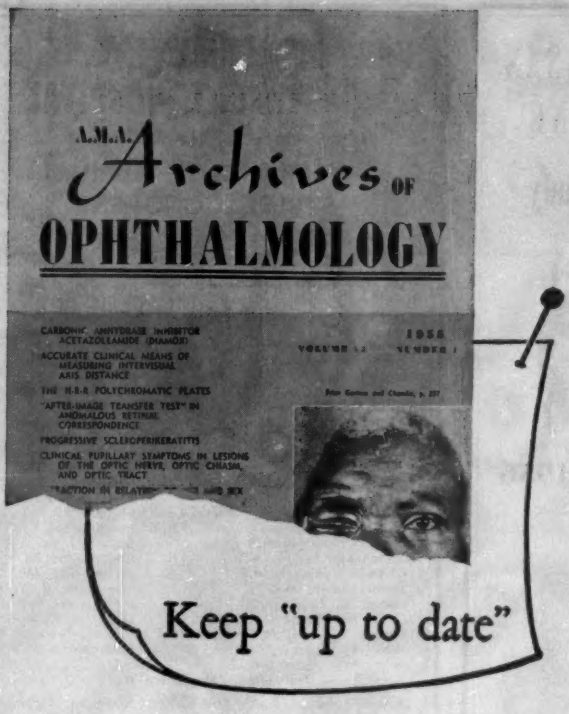
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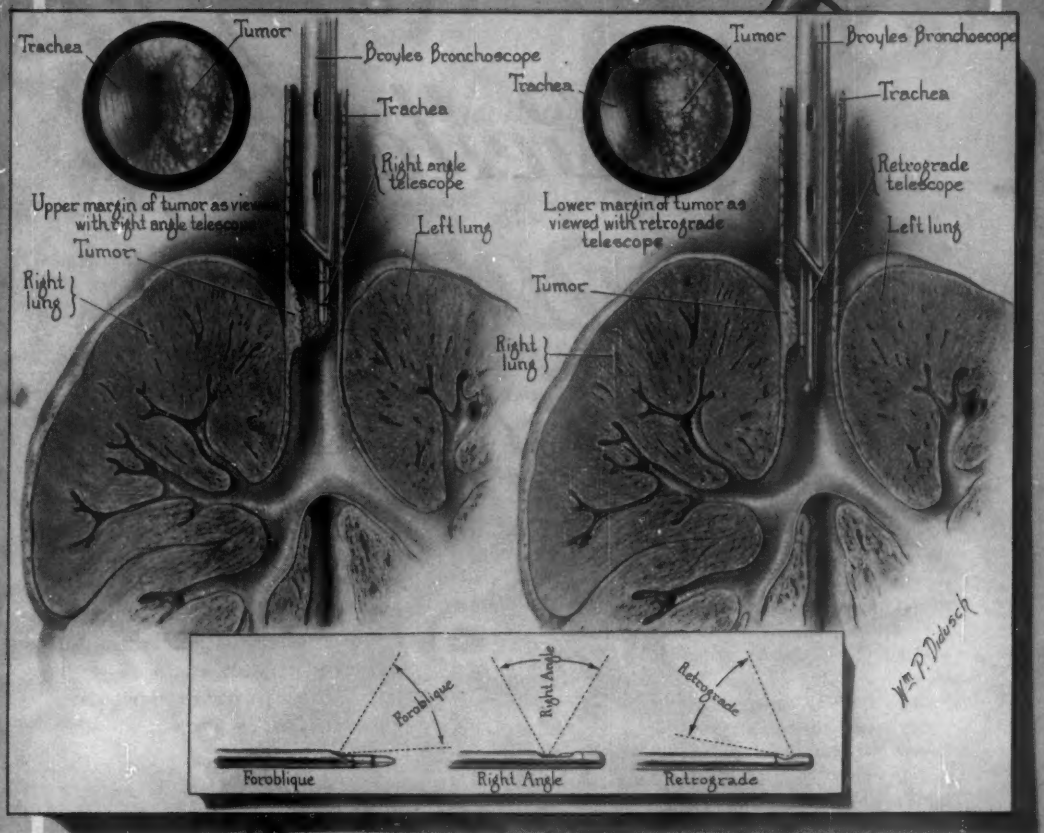

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Reaction to Cocaine Used as Topical Anesthetic in Bronchoscopy

Study of One Thousand Cases

R. A. TELLA, M.D.
and
WILSON WEISEL, M.D., Milwaukee

The necessity for a critical evaluation of the toxicity of local anesthetic agents used in endoscopy was previously expressed by us.[1] As a series parallel to and comparable with the previous group of patients reviewed, in whom tetracaine hydrochloride was utilized as the anesthetic agent, 1000 consecutive diagnostic bronchoscopies on 956 adult patients in whom cocaine hydrochloride was used to induce anesthesia were similarly studied from January, 1950, to December, 1954.

METHODS AND RESULTS OF STUDY

As in the previous study, each of the patients herein reviewed was carefully evaluated before endoscopy by clinical methods, and these data were summarized on special "bronchoscopy work-up sheets." In addition, each patient underwent preoperative skin-testing by intradermal 0.1 cc. wheal injections of 1:1000 cocaine, 1:1000 tetracaine, and isotonic saline solutions, and the results of the three injections were read in 20 minutes and recorded on the work-up sheets; recordings of the blood pressure, pulse rate, and respiratory rate were also made about 24 hours before endoscopy. Premedication consisted of phenobarbital, 128 mg. given hypodermically two hours and one hour before surgery. In addition, meperidine hydrochloride (50 mg.) was usually given 15 to 30 minutes prior to endoscopy.

Anesthesia was secured by placing cotton pledgets, moistened by dipping in 2 cc. of 10% cocaine solution in a medicine glass and pressed dry, between the faucial pillars and in the piriform sinuses for about 60 seconds. Then, 4 cc. of 4% cocaine was placed in the trachea in divided doses, with use of a laryngeal delivery tube and mirror. The exact amount of anesthetic actually used has been almost impossible to determine, but, in general, it can be estimated from gravimetric measurements of the solutions returned that an average of 180 mg. was utilized.

In order to analyze the general effect of cocaine, 100 consecutive patients were studied by serial determinations of blood pressure, pulse rate, and respiratory rate at five-minute intervals before, during, and after anesthetization. When untoward reactions were encountered or suspected, the patient was immediately observed, treated, and simultaneously studied by a special team to record serial determinations of the vital signs as described.

For assessment of the intradermal tests, a wheal of 1 cm. in diameter persisting 20 minutes after administration when the saline wheal had practically disappeared was considered a positive reaction. There were 74 patients in this group who exhibited positive reactions to cocaine. Of this group, 52 patients also showed a similar reaction to tetracaine. Only one of these patients with a positive skin test displayed an untoward

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From the Departments of Anesthesia and Surgery, Veterans Administration Hospital, Wood, Wis., and the Marquette University School of Medicine.

reaction to the anesthetic administration, and none of the other positive reactors developed allergic phenomena of either general or local nature after the procedure. There were 30 other patients who showed a positive skin reaction to tetracaine and in whom the cocaine test was negative.

A survey of the 100 patients studied consecutively revealed that there was an average elevation of blood pressure during the anesthetic procedure of 18 mm. of mercury systolic and 7 mm. diastolic. There were increases in the pulse and respiratory rates which averaged 15 and 5, respectively. In the patients showing the most marked changes, there was definite association with the apprehension or cough and gag reflex stimulation exhibited by the patient. The changes described usually persisted for 15 minutes following the endoscopy.

There were nine anesthetic reactions noted in this group of patients. Seven of these were of a syncopal type and were considered minor, while two were convulsive in character and were considered severe. All these reactions occurred in men and during or immediately after completion of the anesthetic administration. They all started in a similar manner. The patient showed a loss of contact with environment and did not respond to spoken requests. The pupils dilated, and loss of consciousness occurred. The pulse rate became rapid and thready, and the blood pressure dropped. In the two patients with convulsions, the latter appeared simultaneously with the syncope; they started as clonic movements of the hands and feet, spread over the body, and were associated with apparent laryngospasm, breath-holding, and cyanosis until sufficient intravenous thiopental sodium was given to cause muscular relaxation. In the two patients with convulsions, pulse and blood pressure determinations were difficult to obtain until the reaction was controlled and then remained below normal for about three hours.

It is worthy of note that in this series of cases four other patients developed convulsive-like states, but they were not considered to have anesthetic reactions. Two of these patients had hyperventilation syndromes, and the symptoms and signs were reproduced by hyperventilation. One patient who developed peculiar generalized athetoid movements which persisted for 45 minutes was later found to have an abnormal electroencephalogram. One patient who was a known epileptic and diabetic was considered to have had a mild seizure during the anesthetic procedure.

A short summary of the cases in which the anesthetic reactions were noted follows:

REPORT OF CASES

CASE 1.—A 28-year-old white man with far-advanced pulmonary tuberculosis underwent bronchoscopy on Nov. 28, 1950, with topical anesthesia induced by 2% tetracaine without untoward reaction. An acute ulcerative tuberculous endobronchitis was found at that time.

Bronchoscopy was repeated on Jan. 30, 1951, with cocaine solutions, as described, used for anesthesia. On completion of anesthetization the patient became noticeably nervous and jittery. Fine tremors were present in the fingers; dilatation of the pupils occurred, and the patient became unconscious for a few seconds. He was placed supine on the operating table and observed. His blood pressure was 70/30; the pulse rate, 140, and the respiration rate, 14; preoperatively these studies had been 98/70, 92, and 24, respectively. The syncopal reaction was short-lived, and the cardiovascular signs were within normal limits within 20 minutes, after which time bronchoscopy was carried out. In this patient, however, the speech remained thick and incoherent for three hours after anesthesia. The skin reactions to the anesthetic drugs in this case had been negative.

CASE 2.—A 38-year-old white man underwent bronchoscopy on March 7, 1951, because of far-advanced active cavitary pulmonary tuberculosis. Within a few moments after the completion of anesthetization, it was noted that the patient was unresponsive. The pupils were dilated, and the pulse was weak and thready. The patient was placed supine on an operating table, and the blood pressure was found to be 80/40; the pulse numbered 140, and the respirations, 16. Normal determinations on this patient had been 132/90, 84, and 20, respectively.

The patient was studied by serial determinations of the vital signs and observed as to his mental reactions. After 40 minutes, he seemed mentally clear and the blood pressure was 100/70, pulse rate 110, and respiration rate 20. Bronchoscopy was carried out, and no untoward effects were noted. The skin reactions to the anesthetic drugs had been negative.

CASE 3.—A 32-year-old white man was subjected to bronchoscopy on May 22, 1951, because of far-advanced bilateral pulmonary tuberculosis. The skin reactions to cocaine and tetracaine had been positive. The saline wheal had been 0.3 cm. in diameter; the tetracaine wheal, 1.0 cm., with 2.0 cm. of erythema, and the cocaine wheal 1.0 cm., with 2.0 cm. of erythema, 20 minutes after administration.

About three minutes after completion of anesthetization, the patient was noted to be unresponsive with dilated pupils. He was placed supine on an operating room cart. The blood pressure was 70/50. The pulse was palpated at 116, and the respirations were counted at 24. In the ensuing 10 minutes the blood pressure rose to 120/80, the pulse was 92, and respirations numbered 20. He became responsive, and the pupils were of normal size. After another 15 minutes, no untoward effects were noted and bronchoscopy was completed.

CASE 4.—A 40-year-old white man underwent bronchoscopy on Aug. 21, 1951, because of a right upper lobe cystic lesion suspected to be due to coccidioidomycosis. Preoperative skin tests with cocaine and tetracaine solutions were negative; the blood pressure was 130/70; the pulse rate was 88, and respirations numbered 20.

On completion of anesthetization, the patient complained of feeling "woozy." It was noted that he was pale, his pupils were dilated, and his eyes showed a lateral nystagmoid motion. He was placed supine on the operating table, and he did not respond to spoken requests for three minutes. The blood pressure during this period was 80/60; the pulse rate 110, and the respiration rate, 16. He recovered in about 15 minutes and showed no untoward results of the episode.

CASE 5.—A 58-year-old white man was subjected to bronchoscopy on Dec. 11, 1951, because of an arteriovenous fistula of the right lung, associated with familial telangiectasis. On completion of anesthetization, the patient complained of feeling faint; he rapidly became unresponsive and had a syncopal episode. His pupils were markedly dilated but responded to the light reflex. He was placed supine on the operating table, and the blood pressure was 86/60; pulse rate, 82, and respiration rate, 14. This patient's pulse and respiratory rates remained low, and the blood pressure returned to a preoperative level of 116/80 in 20 minutes. He became responsive in 10 minutes, and his pupils returned to normal size in 5 minutes. Bronchoscopy and esophagoscopy were then performed. No untoward reactions were noted after the endoscopy.

CASE 6.—A 68-year-old white man underwent bronchoscopy on Dec. 2, 1952, for evaluation of a probable carcinoma of the right upper lobe. The skin reactions to local anesthetics had been negative. Immediately after completion of anesthetization, the patient had a severe syncopal episode and slumped forward in the anesthetic chair. He was placed supine on the operating table, and it was noted that his pupils were dilated; his pulse was weak and thready, and respirations were extremely slow and shallow.

The blood pressure was 90/70, the pulse 112, and the respiration rate 14. The picture persisted for about 10 minutes, during which period the patient became conscious and the vital signs gradually returned to the preoperative level of 120/80 for the blood pressure, 88 for the pulse, and 20 for the respiration rate. Bronchoscopy was performed, and no untoward effects of the reaction were noted.

CASE 7.—A 40-year-old Negro man was subjected to bronchoscopy on June 9, 1953, because of suspected bronchiectasis. Skin tests with the local anesthetics were negative. After the completion of anesthetization, the patient was noted to be mentally confused, his skin became gray, and the pupils were noted to be dilated. He was placed supine on an operating table, and the blood pressure was found to be 90/60 and the pulse rate 116; the respirations numbered 22. Within 15 minutes, the blood pressure had returned to the preoperative level of 120/80, the pulse was 92, and the respirations were 20. Endoscopy was performed, since the patient was cooperative, but he remained mentally confused with some slurring of his speech for about three hours after anesthesia. Complete physical examination and neurologic survey on the following day showed no unusual residuals as a result of the anesthetic reaction.

CASE 8.—A 67-year-old white man underwent bronchoscopy on Sept. 22, 1953, because of a carcinoma of the left lung. Skin reactions to cocaine solution had been negative, although the reaction to tetracaine had been positive. Immediately after completion of anesthetization, the patient did not respond to spoken requests and a tremor was noted in his hands. He was placed on an operating table, and clonic convulsions involving the arms, legs, and face rapidly ensued. The patient was cyanotic and in a position of opisthotonos. At this time, the pulse rate was 112, the blood pressure was probably 150/100, and there was no obvious respiratory movement.

The patient was given 4 cc. of 2.5% thiopental sodium intravenously, and the convulsions stopped at once. Voluntary respirations began at a rate of 24 and dropped to 20 within a few moments. The blood pressure also returned to a level below the preoperative determination of 140/80, and the pulse rate was 92 per minute five minutes after administration of the thiopental sodium. Bronchoscopy was performed, and biopsy of an oat-cell carcinoma performed. Recovery from the procedure and anesthetic was uneventful, and no residuals of the untoward reaction could be determined.

CASE 9.—A 64-year-old white man was subjected to bronchoscopy on Oct. 29, 1953, because of pulmonary tuberculosis. Skin tests with the anesthetic drugs had been negative. After 1 cc. of the 4% cocaine solution was placed in the trachea and before the anesthetization had been completed, the patient's pupils were noted to dilate and he slumped forward in the anesthetic chair. He was immediately placed supine on an operating table; the blood pressure was 80/50; the pulse rate was 110, and respirations numbered 26. It was noted that a gross tremor had appeared in the fingers, and the patient did not respond. The hand tremor gradually became a course clonic movement which eventually involved both arms, and the blood pressure, although difficult to obtain, was rising and thought to be 150/90, while the pulse remained the same although the respirations dropped to 10 per minute.

The patient was given 4 cc. of 2.5% thiopental sodium solution, and there was an immediate cessation of the convulsions. The respirations were normal at 20, with the pulse rate 92 and the blood pressure 120/70. Bronchoscopy was performed, and the patient's recovery was uneventful and uncomplicated.

COMMENT

Even in defending the use of cocaine anesthesia, some authors * have admitted the rare occurrence of serious reactions, but we have been impressed by the lack of careful individual case study in spite of the large volume of surgical procedures reported in the analyses of such data. Other authors have reported isolated instances and groups of cases showing untoward reactions to cocaine, as well as some fatalities.[4] We have tried by careful evaluation of individual consecutive patients to accumulate information of physiologic and statistical value in assessing this problem of the local anesthetic reaction.

The similarity of the response to cocaine noted in these patients to the reactions observed in a comparable group of patients anesthetized with tetracaine has been rather striking. In both groups of patients studied for cardiovascular effect of the drug, there was a moderate elevation of blood pressure, pulse rate, and respiratory rate, which we have concluded to be independent of the anesthetic and more dependent on patient apprehension and reaction to the procedure.

* References 2 and 3.

The untoward reactions to cocaine we have grouped in two classes: the syncopal or collapse syndrome and the convulsive reaction. In this respect, the reactions in these patients were also similar to the untoward responses observed with tetracaine [1] and to the experimental effects of cocaine described so adequately by Steinhaus.† The incidence of toxic reactions has been significantly lower in this series of patients with cocaine anesthesia than in the group studied with the use of 2% tetracaine,[1] and this may well be corroborative of previous clinical and experimental studies.‡ In addition to the objective studies on these patients, an attempt was made to evaluate the efficacy of anesthesia obtained. It was our impression that whereas anesthesia with cocaine was adequate for endoscopy it was not as effective as that obtained with 2% tetracaine in respect to duration of anesthesia and the abolition of the cough and gag reflex. This impression was furthered by interviews with patients who were questioned subsequent to bronchoscopy after cocaine anesthesia when previous endoscopy had been performed with the utilization of tetracaine.

The actual mode of physiologic action of the anesthetic agent in the cocaine reaction has been a difficult problem to assess completely. We have agreed with previous authors § that the important factors involved have been total dosage of the agent, susceptibility of the patient, and an absorption factor which may or may not be related to vasoconstriction and/or vasodilation. In respect to total dosage, however, it was felt that in this particular group of cases the actual dosage of the anesthetic agent was fairly well controlled by the careful limiting of the amount of anesthetic agent made available for use. In that respect, therefore, it has been assumed that dosage has been constant for all the patierts concerned. As far as susceptibility of the individual patient can be considered, if this is related to an allergic phenomenon which can be defined by the

† References 5 and 6.

‡ References 7-10.

§ References 5, 9, and 10.

usual methods of skin hypersensitivity, we believe that this particular factor can be ruled out in the present group of cases, since the skin-testing showed no relationship between positive reactors and the so-called anesthetic reactions. This leaves, then, for final consideration the problem of absorption of the anesthetic, as well as its rate of absorption, as the remaining factor which cannot be answered by our study, although a project is under way at present in an attempt to determine the actual effect of this phenomenon in respect to the local anesthetic modality.

The best treatment for the anesthetic reactions would be their prevention, and we have attempted to accomplish this by the careful method of anesthetic administration described and the preoperative use of barbiturates. The factors enumerated, we believe, are effective, but when untoward reactions occur in spite of these precautions, the use of intravenous thiopental sodium is necessary to control the severe convulsive type of response. In addition, complete anesthetic and operating room facilities should be available in the management of these patients. For the minor type of anesthetic reaction of a syncopal or collapse variety, we believe that simple observation or the utilization of Trendelenburg position is all that is necessary. In the event, however, that there is any question in the mind of the surgeon about the seriousness of the anesthetic reaction, we feel it is wiser to give the patient intravenous barbiturates, and in our hands thiopental sodium has proved to be satisfactory.

SUMMARY AND CONCLUSIONS

A review of 1000 consecutive bronchoscopies performed on 956 adult patients with the use of topical cocaine anesthesia is described, with careful evaluation of each individual patient. In this group of patients there were seven minor and two severe anesthetic reactions.

The anesthetic reaction found in the use of cocaine has been of two principal types: a minor or syncopal type of reaction and a severe or convulsive response.

The use of cocaine has produced fewer toxic reactions than the use of tetracaine anesthesia. The fact that reactions of toxic nature do occur with cocaine, however, makes it imperative that provision for management of these reactions be immediately available in the operating room. For that reason, it is felt that endoscopy should be performed in a hospital where adequate preoperative medications can be utilized and where resuscitative measures are immediately available in the endoscopy room.

The factors of anesthetic absorption appear to be the most important problem involved in occurrence of anesthetic reactions.

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Wegener's Granulomatosis

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Wegener's granulomatosis is an unusual syndrome that anatomically and pathologically is characterized by necrotizing granulomatous lesions in the respiratory tract, generalized necrotizing angiitis, and glomerulonephritis, and it is of special interest to the otolaryngologist. Several recent articles * give excellent discussions of the syndrome, together with accounts of new and previously reported cases that fulfilled the diagnostic criteria. Klinger,[4] in 1931, reported what is now considered to be the first recorded case of the syndrome, and Wegener,† in 1939, reported three cases and postulated the syndrome as being a disease entity. There is disagreement as to what constitutes the entity, i. e., whether nasal lesions are pathognomonic of the entity ‡ or whether involvement of an unspecified part of the respiratory system is distinctive.[1]

In this paper one case that I believe fits the requirements for the diagnosis of Wegener's syndrome is briefly presented, together with important features of the syndrome, and a case is reported that clinically resembled Wegener's granulomatosis but on postmortem examination did not fulfill the diagnostic criteria.

REPORT OF CASES

CASE 1.—A 51-year-old Negro woman was first seen as an outpatient in July, 1954, with presenting complaints of upper abdominal pain and epigastric burning sensations. Pain also had been noted on deep breathing. Five years earlier the patient had had a duodenal ulcer and two months before examination she had had one episode of painless hematuria. During the three weeks prior to examination, her temperature had been consistently elevated, occasionally to as high as 101 F; temperature elevation was associated with weakness of the hands and arms and swelling of the joints in both hands. Past history revealed frequent colds for approximately 13 years, and growths, presumably nasal polyps, had been removed 10 years previously. Family history revealed that one of the patient's brothers had had pulmonary tuberculosis.

A roentgenogram of the chest showed what appeared to be bilateral pleural thickening with possible effusion, but a pleural tap was not diagnostic. A gastrointestinal series also was not diagnostic. Roentgenograms of the spine showed cervical and thoracic osteoarthritis. A preliminary diagnosis of tuberculous pleuritis was made, and treatment with streptomycin and aminosalicylic acid was given for approximately six months.

Shortly after the discontinuance of streptomycin administration, the patient was seen for the first time in the department of otolaryngology because of the chief complaints of diminished hearing and tinnitus of about one month's duration; she also had had headaches and thick mucoid postnasal discharge. The nasal examination revealed an irregular growth in the left middle meatus, which was covered by a crust and bled easily on manipulation. A biopsy specimen of the growth was reported as representing chronic granulomatous inflammation, described as follows: Areas of infiltration by histiocytes with slight or no focal arrangement and with rare giant cells, no multinucleate cells of the so-called foreign-body type. Lymphocytic infiltration was present marginally, and outside of this some plasma cells and neutrophiles were observed. No Reed-Sternberg cells were found. Gridley and acid-fast stains revealed no organisms. Roentgenograms of the sinuses showed only a slightly thickened antral mucosa. The loss of hearing was a perceptive type of impairment, and the streptomycin was considered to have been the probable causative agent.

Approximately one month later, she again was examined, because of a recurrence of her original symptoms: pain in the left side of the chest, generalized weakness, and aching of the body, particularly in the arms and knees. Her temperature was

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* References 1-3.

† References 5 and 6.

‡ References 3, 5, and 6.

100.2 F. She was admitted to the hospital for further study. A repeat roentgenogram of the chest showed a slight decrease in the pleuritic reaction. There had been no progression of the nasal lesion, and a repeat biopsy still showed chronic granulomatous inflammation. Biopsy of the gastrocnemius muscle revealed necrotizing arteriolitis. On the basis of the findings from these biopsies, the pathologist's tentative diagnosis was Wegener's granulomatosis. Essential findings on urinalysis at that time were a trace of albumin and occasional red blood cells and white blood cells. Essential findings in the blood analysis were as follows: white blood cell count, 7200/cu. mm.; hemoglobin, 10.5 gm/100 ml., and cell volume, 39 ml/kg. body weight, while the differential count showed 2% eosinophiles. Urine and blood cultures were reported as showing no growths.

The treatment consisted of a course of nitrogen mustard (methyl-*bis* [β-chloroethyl] amine hydrochloride), isoniazid, and pyridoxine. When the patient was last seen, on March 28, 1955, at the time of her discharge from the hospital, she was asymptomatic so far as chest pain and generalized weakness and aching were concerned. The temperature for the week preceding her discharge had been normal.

CASE 2.—A 54-year-old white man was first seen in March, 1952, with a presenting complaint of aching in the dorsal part of his chest. Neuromuscular pain was diagnosed. When he returned two years later, in March, 1954, he stated that the aching had improved but that he intermittently had a sensation of needling in the dorsal part of the chest. A roentgenogram of the chest showed no active pulmonary lesion.

About six months later, the patient returned with complaints of constipation, chronic nonproductive cough, soreness of the left eye, and loss of weight of about 9 lb. Ophthalmologic examination revealed a granular lesion of the left conjunctiva. A biopsy specimen was reported as showing chronic inflammation. A roentgenogram of the chest demonstrated an area of infiltration at the base of the upper lobe of the right lung. Bronchoscopy revealed a granular area near the orifice of the upper lobe of the right lung. The pathologic diagnosis of a biopsy specimen from the granular area was chronic inflammation and granulation tissue. Because of these findings and the short duration of the symptoms, an exploratory thoracotomy was performed; a mass was found which grossly appeared to be neoplastic. A right pneumonectomy was then performed. The pathologic diagnosis was chronic suppurative and granulomatous pneumonitis, with marked fibrosis and focal carnification, suggesting the possibility of Pasteurella infection. Agglutinations for tularemia and brucellosis were negative, as was a culture of the lung tissue. The gastrointestinal symptoms were undiagnosed, as the results of all studies, including a gastrointestinal series, were negative.

Two months later, in November, 1954, the patient was seen for the first time in the Department of Otolaryngology, with a complaint of rawness inside the nose of about one month's duration. Examination revealed moderate crusting in both nasal cavities, and the mucosa bled easily on gentle manipulation. The turbinates were moderately atrophic.

The patient was not examined again until April, 1955. He had been in California, where he had had to be hospitalized because of recurrence of cough and bloody sputum. The patient had become emaciated and extremely weak, and a pronounced saddle deformity of the external nose was now apparent. There was marked crusting within the nasal cavity; also several perforations of the nasal septum had developed since previous examination. The patient appeared to be terminally ill.

Roentgenograms of the chest showed several new patchy infiltrated areas in the left lung; the right pleural space was obliterated. Roentgenograms of the paranasal sinuses showed clouding of all sinuses. Urinalysis revealed one to three red blood cells and an occasional pus cell per high-power field. The white blood cell count was 14,500/cu. mm. The diagnosis of Wegener's granulomatosis was considered; treatment consisting of nitrogen mustard empirically was begun. The patient died within five days.

Postmortem examination showed granulomatous lesions in the nose, nasopharynx, larynx, trachea, and left lung. The kidneys revealed glomerulonephritis but, as in the other histopathologic sections, blood vessels did not appear to be involved; in no part was there evidence of a necrotizing angiitis. Blood cultures were reported as showing no growth.

COMMENT

Patients with Wegener's granulomatosis may have the presenting symptoms of chronic rhinitis, sinusitis, otitis media, chronic cough, hemoptysis, or unresolved pneumonia. Occasionally, constitutional symptoms, such as fever, malaise, or loss of weight, may be the complaints, and lesions are revealed only after otolaryngological, pulmonary, or roentgenographic examinations. If the lesions are located in the nasal passages, the usual chemotherapeutic agents are ineffectual in halting cartilaginous and body destruction, which may cause saddle nose, proptosis, or exophthalmos. The lesions may also extend downward and establish a nasal-oral fistula.

The first case reported here appears to be one of Wegener's granulomatosis, showing some tendency toward remissions. The patient's symptoms and condition corresponded to the accepted criteria for diagnosis, although the renal findings so far are minimal. In other reported cases,§ the patient usually is febrile, and anemia, leucocytosis, and elevated sedimentation rates commonly are found. Blood serology is negative, and lymphadenopathy and hepatomegaly have not been observed. Renal damage usually occurs in the later stages, the signs varying from moderate albuminuria with few red cells to frank hematuria and oliguria. The interval between the first appearance of renal damage and death may be as brief as 10 days. No organisms have been identified as causative agents. Remissions have been noted during chemotherapy,[1] which probably result from control of secondary infection; spontaneous temporary subsidence of symptoms has been noted in some cases.[1]

The second case is more difficult to classify; it differs from the typical midline granuloma (which is localized to the nasal region) in that the lesions did not remain localized and from Wegener's granulomatosis (in which necrotizing angiitis is present) in that necrotizing angiitis was absent. Thus, the case was classified as an idiopathic midline granuloma involving nose, nasopharynx, larynx, and pulmonary tract.

Differential Diagnosis.—The lesions of Wegener's granulomatosis can be confused with the idiopathic lethal midline granulomas,‖ although the latter usually remain localized and show no evidence of vascular or renal involvement. The lesions of the reported cases, i. e., Case 1 of Woodburn and Harris[10] and Case 3 of Williams,[9] fit the criteria, and these cases should be considered as being cases of Wegener's granulomatosis. At times the lesions may be suggestive of specific infectious granulomatous diseases, such as tuberculosis, syphilis, blastomycosis, tularemia, or glanders; but in these diseases, in contradistinction to Wegener's granulomatosis, the causal organism usually can be recovered and generalized necrotizing lesions are absent. Mycosis fungoides or neoplastic disease must be considered in the differential diagnosis; however, biopsy and absence of generalized necrotizing lesions should eliminate these. Atrophic rhinitis may be suspected; it is distinguished by its relatively long duration and the lack of progressive destruction. Sarcoidosis may cause granulomatous nasal lesions and involve similar areas,[11] but the lesions are not necrotizing and do not cause necrotizing glomerulitis.

Lesions in the lower respiratory tract may involve any portion of the lung. Findings on physical examination of patients having Wegener's granulomatosis may be unremarkable or may indicate pleuritic or pneumonic processes. Roentgenograms of the chest may give the appearance of tuberculosis, neoplasm, or nonspecific parenchymal infiltrations. The pulmonary findings in Löffler's syndrome[12] simulate those in Wegener's, but the vascular lesions outside the lungs in Löffler's syndrome are not generalized. The pulmonary lesions of Wegener's syndrome (as well as the nasal lesions) also do not respond to chemotherapy.

Other areas sometimes are involved in Wegener's granulomatosis; episodes of arthritis, neuritis, carditis, parotitis, and prostatitis have been recorded.[1]

Wegener's granulomatosis has been considered variously to be a respiratory renal subtype of periarteritis nodosa,[2] hypersensitivity angiitis,[13] or the microscopic form of periarteritis nodosa.[14] However, Fahey and associates[1] differentiate Wegener's granulomatosis from those conditions by the considerable variation in vasculitis (pulmonary vasculitis being uncommon in typical periarteritis nodosa) and the appearance in Wegener's granulomatosis of the extensive necrotizing granulomatous lesions concentrated in the respiratory tract preceding the onset of generalized vascular disease. Churg and Strauss[15] describe a syndrome as aller-

§ References 1 and 3.

‖ References 7 and 10.

gic angiitis and granulomatosis, but differentiation has been made [1] on the basis of the following findings in Wegener's syndrome: (1) the progressive character and marked predominance of the necrotizing lesions in the respiratory tract; (2) the regular occurrence and severity of renal lesions, and (3) the absence of a history of allergy or of tissue eosinophilia (although the presence of either of these does not necessarily preclude the diagnosis of Wegener's syndrome).

We are in agreement with Fahey and associates,[1] who believe that some relationship exists between hypersensitivity states and Wegener's syndrome, because of the coexistence in each of granulomatosis, vasculitis, and glomerulonephritis.

SUMMARY AND CONCLUSION

Wegener's granulomatosis is an uncommon disease that is of interest to the otolaryngologist. Symptoms pertinent to differential diagnosis are discussed, and two cases are presented: one that is believed to satisfy the diagnostic criteria of Wegener's granulomatosis; the other that was similar but illustrates idiopathic midline granuloma involving nose, nasopharynx, larynx, and pulmonary tract. The latter case differed from the typically described midline granuloma in that the lesion did not remain localized to the nasal region and differed from Wegener's syndrome in that necrotizing angiitis was absent. The etiological factors are not known in these diseases; perhaps all these diseases represent different stages of the same disease.

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Gross Pathology of Sinus Discharge

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The use of the naked eye in pathologic diagnosis is routine and has been since the early days of medicine. The application of this routine to study of the sinus discharge readily yields information of value which other methods fail to uncover. This information concerns the impairment of function of the lining membranes, the loss of resistance to infection, the loss of power to evacuate the sinus, and the persistence of purulent characteristics with a variety of appearance, all of which indicate persistent pathology within the sinus and the probable presence of infection.

While pus-forming bacteria, when present, are readily detected in sinus discharge by common laboratory technique, it is not always possible to determine their significance in a given case. Dochez [1] and others have demonstrated the importance of viral agents in nasal pathology, and many workers have improved the cultural methods and means of identifying these agents. But here again, to determine their presence and significance in sinus infection is beyond the reach of the usual practice in otolaryngology. It is possible, however, to determine by a study of the gross characteristics of sinus discharge the degree of injury the lining membrane of a sinus has received from infection and the probability of this membrane returning to a normal condition and remaining so. The demonstration of pathology within a sinus and loss of function of the lining membrane increases the likelihood of their being factors in systemic disease.

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The diagnostic value of transillumination and x-ray is considerable, but the most accurate method of determining the vitality of the lining membrane and the chronicity of sinusitis is found in the study of the discharge returned from irrigation of the sinus. Such a study reveals the persistence of the inflammatory condition and the degree of regression that has taken place within the sinus. A sinusitis that is not reversible to a normal state is readily determined.

Nasal accessory sinus inflammation affects the air-filled cavities connecting to the nasal chamber by a small natural opening and lined by a columnar ciliated mucous membrane containing mucous cells and numerous mucus-secreting glands.[2] Normally, the air is moist, as in the nasal chamber, and there is only a slight trace of clear secretion. The reaction to pathologic stimuli depends upon the severity of the stimulus or injury and the duration of it.

The maxillary sinus is so situated that discharge is readily obtained from it by direct irrigation. This irrigation can be done under local anesthesia by puncture through the inferior meatus with a trocar or by irrigation through the natural opening after cannulization. The puncture method is the method of choice because, while it is a somewhat more rugged procedure, the sinus cavity is irrigated from below, out through the natural opening, and so can be completely evacuated of whatever discharge is present.

The routine technique used for obtaining this discharge involves cocainization of the inferior meatus and the nasomaxillary wall. Cleansing of the nasal chamber is then done with 0.5% sterile sodium bicarbonate solution, using the suction irrigation method. A

trocar is then introduced into the maxillary sinus by puncture through the usual site beneath the inferior turbinate. The solution used in each sinus is 200 cc. of isotonic saline sterilized by autoclave, followed by 20 cc. of tyrothricin, 1 : 5000, or other antibacterial solution. The trocar, metal syringe, and utensils are sterilized by boiling. The returned wash is collected in clean sterile basins and then placed in sedimentation glasses. This gives an adequate opportunity not only to obtain discharge directly from the sinus but to observe the character of the discharge accurately by transmitted light. Blood cloud arising from the puncture is cleared by a few drops of dilute hydrochloric acid.

Normal sinus secretion is minute in quantity and consists of one or two very small shreds of clear glassy material evidently composed of mucus. This should be expected from the normal anatomy of the lining membrane, which has not only mucus-producing columnar epithelium but also mucus-secreting glands. The normal secretion is important in keeping the lining membrane moist, thus giving the cilia of the lining cells a proper medium in which to maintain their mobility. The secretion then does not stagnate but moves constantly toward the natural opening.

The production of discharge within the cavity starts when the infection enters the sinus cavity. It is well accepted that this occurs through the natural opening of the maxillary sinus by extension from the nasal chamber.[8] Since the anatomical elements of the lining membranes are largely similar to those of the nasal membranes, the discharge there parallels that of the nasal chamber. At first, it is thin, clear mucus, becoming thicker and then more purulent according to the severity of the inflammatory process. The subsequent character of the discharge within the sinus cavity depends upon the severity of the infection and the degree of injury which the lining membrane receives. Any collection of discharge of more than the normal few clear mucus shreds indicates the impairment of function of the ciliary mechanism of the lining of the sinus and inability to evacuate the sinus.

The mucus content of the discharge is extremely important in determining the degree of injury to the membrane. Mucus binds the discharge together into clumps and rolled-up masses readily discerned on stirring. The purulent content of these rolled-up masses of mucus is easily seen. This varies from a ground-glass appearance to one of solid pus. On stirring, these masses of discharge do not break up unless the mucous element which binds them together is absent. When the mucus no longer binds the discharge into masses, injury to the membrane is severe and involves the deep structures of the membrane which contain the mucus-secreting glands. When the inflammatory process is of short duration, the discharge tends to be large in quantity. In processes of longer duration the discharge may also be large, but this group often has discharge that is scanty.

The change in character of the returned discharge as the condition improves clinically is readily seen. As the washing of the maxillary sinus is continued over a period of time, the amount of permanent damage can be estimated from the amount of impairment of the mucus-forming elements as shown in the recovered wash. While the quantity of discharge may be constantly less, the persistence of purulent elements in this discharge indicates continued pathology. Even when the return is scanty, but the shreds of returned discharge are stained with purulent material, this conclusion can be made.

When there is complete loss of function of the mucus-forming elements of the lining membrane, the discharge is completely purulent. Under these conditions a considerable variation in quantity, consistency, and appearance is seen. The factor of the sinus being an air chamber with a drying effect may be manifest. The returned discharge may contain a collection of fine, dry, scabby purulent material, or it may be milky in appearance. The presence of a faint cloud

in the wash is often difficult to see, but, after the wash stands in the sedimentation glass for an hour or so, an appreciable amount of purulent material settles to the bottom, where it is easily seen. When this is stirred, the cloud again forms. Occasionally, a thin, almost watery, yellowish cystic type of fluid is returned. It is interesting to note that Hurd,[4] in an article published in 1930, pointed out that a sinus discharge which is milky in character indicates serious damage to the mucosa. Blood-stained discharge occurs occasionally in acute inflammation of the sinus. It indicates here a severe type of injury to the lining membrane. In chronic conditions it indicates ulceration of the membrane and probable malignancy.

Return to normal of the sinus membrane is shown by the progressive loss of purulent characteristics of the recovered discharge, followed by the reappearance of normal mucus that is normal in amount. The ciliary action is now functioning to move the secretion. Many cases in which the discharge is present several weeks following an acute episode will show a continued reduction in the quantity of discharge, but still the discharge will not be altogether clear mucus. Then, after a rest from treatment, if the discharge returned by wash has now clear mucous shreds, it can be said that the infection is controlled but that the ciliary mechanism is not completely returned to its normal function. On this basis, it can be concluded that there is still an opportunity for the membrane to return to a normal state. When the quiescent period has been passed and the sinuses are then irrigated, with the return still containing a quantity of mucus having purulent elements visible in it, then it can be said not only that the ciliary mechanism is damaged but also that infection is still present.

In the cases in which the discharge returned is always purulent in character and the mucous elements are not present, it is evident not only that the ciliary mechanism is injured but also that the pathologic change involves the mucus-forming elements, which have likewise lost their function. This type of discharge shows serious damage of the lining membrane, and the condition is difficult to clear by treatment because of the deep-seated change present in the sinus.

Modification of the character and quantity of discharge recovered after the first irrigation of the sinus is common. This is manifest in the mucous and purulent components of the discharge, as well as in the quantity of it. Increased discharge following an irrigation of the chronic maxillary sinus is almost the rule. The possibility of this increased discharge being caused by reinfection of the sinus by this procedure is very remote when the sterile technique outlined here is used. The more abundant discharge and changed character must, therefore, be due to the beneficial action of the irrigation and the consequent improvement and revitalization of the lining mucosa and the discharge-forming mechanism. When this reaction occurs, the discharge increases under treatment as the function and vitality of the membrane return. Continued treatment may then bring about an improvement in the character of the discharge returned and a decrease in its amount. But when a reaction of increased discharge is found in a sinus, it is often the case that the original normal resistance of the membrane cannot be restored by irrigation. This reaction is especially evident when the drying effect is present within the sinus and the scabby quality of return is encountered.

Surgery of the maxillary sinus in which a drainage opening is made through the nasomaxillary wall beneath the inferior turbinate gives ready opportunity for extended study of the discharge from the sinuses on irrigation. Here, the steadily improving condition of the lining membrane is often shown by the character and appearance of the discharge returned from irrigation through this opening. It is interesting that this improvement in the character of the discharge is often slow to take place but that the improvement may go on over a period of many months.

SUMMARY

A study of the gross appearance in transmitted light of the discharge recovered from the maxillary sinus by irrigation reveals pathologic changes in the lining membrane of the sinus. An excess amount of discharge indicates damage to the ciliary mechanism. The mucous content reveals the pathologic condition of the mucus-forming elements. Presence of purulent material indicates the existence of infection. The drying effect of the air chamber is an important factor in the appearance of the discharge. Increased discharge following irrigation is due to improved condition of the lining membrane. Reversibility of the damage to the membrane is readily determined by observation over a period of time.

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Atresia of the External Ear Canal

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INTRODUCTION

Atresia of the external ear canal may be congenital or acquired. Congenital atresia presents a more complex problem. If it is bilateral, a severe hearing impairment exists from birth, with consequent tardy development of the child. A speech defect is coexistent. The cosmetic deformity is disturbing to the parents early, and later to the patient. These patients are often neglected. Progress in otological surgery has made correction possible, with considerable improvement in the hearing and in the cosmetic appearance. This paper presents another suggested form of surgical therapy.

HISTORY

Kiesselbach,* in 1883, is credited with the first attempted operation to correct congenital ear deformities. From the literature, it is apparent that misunderstanding of the embryonal development resulted in poor results. Dean and Gittens [2] in 1917 reviewed the various types of operations and reported hearing improvement in their case. In 1935, Hume and Owens [3] reported two cases with hearing improvement. Cohen and Fox,[4] in 1943, reviewed the literature and reported a case of traumatic atresia of the external ear canal.

In 1947, two independent reports gave the most optimistic expectation of consistently good results. Ombredanne [5] described two cases upon which he successfully operated. George Pattee [6] reported five bilateral cases. Vogel † in 1949 reported a case in which fenestration was performed, with marked hearing improvement, but the external canal failed to remain patent. Successful cases were then described by Siirala [7] in 1949 and by De Graaf Woodman [8] in 1952. A good review was presented by House [9] in 1953. He recommended establishing a middle ear air space by allowing the bony eardrum to remain. Theobald,[10] in 1954, described his series of 12 cases.

Aplasia has been suggested as a term to replace congenital atresia. Atresia suggests closure of a canal that previously was patent, whereas aplasia denotes its failure to develop. Common usage, nevertheless, permits congenital atresia to be used to indicate closure of the external ear canal together with defects within the middle ear and of the drum membrane in addition to defects of the pinna.

ETIOLOGY

In order to understand and treat congenital ear canal atresia adequately, an understanding of the development of the ear is essential. The inner ear, or sensory end-organ, develops first, from an invagination of the ectoderm, forming the otocyst. The labyrinth and cochlea develop from the otocyst. Anomalies of the inner ear occur without any associated defects of either the middle or the external ear. For example, a child born deaf with a nerve-type loss from rubella in the mother presents such a deformity. Imperfect nerve connections and faulty development of the cochlea are other examples of congenital deafness.

After the inner ear is fairly well presented, from the gill structures comes the sound-conducting portion of the hearing apparatus, the middle and the external ear. The first

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* Kiesselbach, W., cited by Fraser.[1]

† Vogel, K., cited by House.[9]

pharyngeal pouch differentiates into the Eustachian tube and the tympanic cavity of the middle ear. Within the loose connective tissue of the middle ear, the auditory ossicles develop. The malleus and incus are derived from the dorsal end of the first arch (Meckel's cartilage). The stapes is derived from the second branchial arch (Reichert's cartilage). The malleus attaches to the eardrum, the stapes is inserted into the oval window of the perilymphatic space, and the incus articulates with the other two. We are certain of the branchial origin of the auditory ossicles because of the innervation of the tensor tympani muscle for the malleus from the first arch and the stapedial muscle by the facial nerve from the second arch.

The tympanic drum membrane is formed by the meeting of the first branchial cleft and the first pharyngeal groove. The outer layer of the drum membrane is ectodermal, with a thin layer of mesodermal tissue separating it from the entodermal layer of the middle ear.

The external ear is formed from the first branchial groove together with additions from the nearby branchial arches. The external ear canal represents the ectodermal groove itself. The pinna develops around the first groove by forming six hillocks, which form ultimately the characteristic auricle. Defective combination of the hillocks is responsible for the various types of malformed pinnae. Inhibited development presents a fetal type of pinna.

Deformities of the auditory apparatus may be entirely independent of each other. The external and middle ear may be deformed with a normal inner ear structure. The facial nerve is often stated to be in an abnormal position and may be smaller. The nerve may pass perpendicularly from the dura instead of having the usual bend under the horizontal semicircular canal.

The incidence of congenital atresia is probably about one case per thousand ear, nose, and throat patients. Congenital atresia of the canal without deformity of the pinna is seldom seen. Unilateral atresia is commoner. Males appear to be affected oftener, and the right ear is more frequently affected. With an atresia of the external ear canal, a normal eardrum is believed to occur rarely. In place of the drum membrane is found an occluding plate of bone. According to embryological study, the bone is believed to be formed from the hyoid bone of the second branchial arch. The temporomandibular joint develops in a more dorsal position, in very close association to the mastoid portion of the temporal bone. Frequently scar-tissue bands are present within the tympanic cavity. The malleus and incus are fused, and the malleus may be fused to the bony occluding plate in place of the membranous eardrum. The stapes is normal, but abnormalities of the incudostapedial joint may be present. The Eustachian tube is normal. In the same patient, one must be on the guard for other congenital abnormalities.

Acquired atresia of the ear canal may be caused by soft-tissue or bony disease. The stenosis may be complete or partial. Chronic inflammation, such as eczema, recurrent otitis externa, and long-standing purulent otitis media, may cause changes in the soft tissue. Chemical cauterization for carcinoma or the use of the actual cautery may cause diaphragms, bands, or rings with scarring and complete occlusion.

Endaurally performed mastoidectomy may cause a stenosis in certain persons with keloid tendencies. Atrophy of the cartilage occurs in the aged, with collapse of the canal walls followed by obstruction. Tumors of the external canal, such as osteomas, exostoses, cysts, and malignancies, produce obstruction. Trauma from automobile accidents may cause cicatricial growth within the canal. Burns of the auricle and canal may be followed by stenosis. Involvement of adjacent tissues, such as lymph nodes or parotid gland abscesses, may rupture into the ear canal, producing atresia.

FINDINGS AND DIAGNOSIS

A conduction-type hearing loss is invariably present with obstruction of the ear canal by atresia, either congenital or acquired. It is important to determine the existence of

any cochlear type of loss. One is unable to determine this accurately in congenital atresia before the age of 4 years.

With complete absence of the sound-conducting portion of the hearing apparatus, a loss of 50 to 60 db. is expected. If the phase relationship between the round and oval windows or the fenestra following surgery is active, the hearing can be raised to a 20 to 30 db. loss.

The auricle may demonstrate all degrees of developmental error from complete absence to microtia, to distortion, or to accessory appendages. The external meatus may be absent completely or possess a pit or blind funnel shape of varying depth. At the site of the membrane tympani is usually found a bony plate. Rarely is a normal eardrum found with a completely atretic canal. The incus and malleus are deformed at times, with fusion to the bony plate. Seldom is the stapes abnormal, but it may be fixed because of articulation with the fused ossicles. A fixed deformed stapes may occur if there is an isolated involvement of the second branchial arch (Reichert's cartilage). Characteristic of congenital fixation of the stapes is a history of deafness since birth with nonprogression of the hearing loss.

Associated with atretic ear canals may be faulty development of the mandible, facial nerve paralysis, or hemiatrophy of the facial muscles. Coincident with the Treacher-Collins syndrome is congenital ear atresia with the characteristic aplasia of the jaws, notching of the lower eyelids, with absent puncta and oblique slanting palpebral fissures.

Pneumatization of the mastoid is usually present but may be absent, which may mean that the area of the middle ear cavity is solid bone. Routine roentgenograms of the temporal bone should be taken. Special techniques will reveal the external canal accurately and consistently, and also the ossicles of the middle ear if present. Camp and Allen [11] described a method utilizing tomograms revealing the canal in coronal section and, for confirmations, in sagittal section. Dr. Gilbert R. Owen,[9] through his technique, has revealed presence or absence of the ossicles through a modified Stenver projection.

INDICATIONS FOR SURGERY

Surgical correction of bilateral congenital ear atresia with normal cochlear function and mastoid pneumatization is agreed upon by all experienced surgeons. Patients with acquired complete atresia of the ear canal also should undergo surgery, as existence of a functioning middle ear is certain.

Correction of unilateral congenital atresia can be undertaken for cosmetic reasons if the patient desires. Localization of sound may be improved, but a 20 to 30 db. hearing loss will yet remain even after a perfect result. Danger of a facial nerve paralysis must be considered before surgery is undertaken.

In congenital bilateral atresia, failure of speech development with associated intellectual retardation makes early surgical correction imperative. The age of 4 years is considered by many men to be the best age because an audiogram demonstrating normal cochlear function can then be done. However, labyrinthian turning tests and possibly caloric tests at an early age giving a normal response can indicate by assumption that the cochlea functions well. Therefore, surgery could be undertaken even one to two years earlier.

TECHNIQUE OF SURGERY

Endaural exposure of the congenital atretic ear is best, the incision always anterior to the pinna. Reports of inadvertent cutting of the facial nerve with the initial incision in congenital atresia call for awareness of its possible malposition.

With the electric bone drill, the cortex of the mastoid is removed and the antrum is entered. The lateral semicircular canal can then be identified. Both Pattee [6] and House [9] suggest as small an opening as possible into the middle ear to minimize consequent scarring.

The bony eardrum, with the deformed ossicles, is visualized under magnification.

Mobility should be checked. If fusion of the incus and malleus and possibly also fusion to the bony eardrum cause fixation of the ossicular chain, some men think complete removal is justified. House[9] believes that the bony eardrum should remain to allow an air space, preventing subsequent fibrosis. Mobility of the stapes can be determined. If the stapes is fixed, which is not usual, fenestration may be considered possibly at a second stage, as recommended by De Graaf Woodman.[8] A split-thickness skin graft is obtained. Our procedure at this stage is to obtain a hollow polyethylene tube of appropriate size for the new canal. The skin graft is thinned where distally it will abut against the stapes or bony eardrum, if this is allowed to remain. The graft is wrapped around the lubricated polyethylene tube, fitting snugly against the newly made bony canal. Fine nonabsorable surgical (silk) sutures fix the graft laterally to the skin edges of the external meatal wall. The hollow tube affords excellent drainage and can be allowed to remain indefinitely. Removal for cleaning may be done one week after surgery. Reinsertion is simple after boiling, or another tube may be used. Subsequent stenosis is prevented by continual use of the polyethylene tube for three or more months. The skin-graft take is assured.

Cosmetic correction of the deformed auricle may be undertaken surgically. With ear prostheses readily available, however, a more pleasant appearance is assured.

Surgery is much simplified in acquired atresia. The scar, usually a cone-shaped diaphragm, is excised and, with the electric bone drill, the canal is widened. The middle ear is not exposed. Polyethylene tubing and skin graft, usually obtained from the abdomen or thigh, complete the procedure.

REPORT OF CASES

An infant was seen at 6 months of age, with bilateral complete bony atresia of the ear canals. The pinnae were small and rolled superiorly but not unpleasantly deformed. At 18 months of age, turning tests revealed normal labyrinthian response. The parents were told to make the boy aware of sound. X-rays revealed an air space in the area of the antrum and middle ear bilaterally. At 22 months of age, the right ear was operated on. The malleus was fused to the bony eardrum. Disarticulation of the incus was accomplished by removal of the bony eardrum with the fused malleus. There was apparent mobility of the incus and stapes. The incus did not appear deformed. A skin graft from the thigh was applied over a polyethylene tube, abutting against the incus.

The child is now beginning to speak simple sentences at 30 months of age, whereas before, even at 22 months of age, he appeared unable to speak. The canal has contracted very slightly. The distal end of the graft vibrates with the pneumatic otoscope and also with inflation of the Eustachian tube. An audiogram has not yet been taken. He has since been diagnosed as having cerebral palsy because of poor use of a leg and arm.

Two cases of acquired stenosis were operated on. One patient had carcinoma of the external ear, treated by the Mohs chemosurgical method, with consequent complete atresia of the external ear canal. The electric dental drill was used to remove the posterior-superior wall of the bony canal. A skin graft was obtained from the abdomen and placed in the area over a polyethylene tubing of appropriate size. The tubing was allowed to remain for three months, with removal at short intervals for cleansing. An excellent result was obtained.

Another case consisted of stenosis following an automobile accident, with extensive lacerations of the side of the face involving the external meatus. A complete closure of the external ear canal resulted. The scar was a cone-shaped diaphragm extending into the inner third of the external canal. At surgery the scar was excised by dissection. The bony canal was widened with the electric drill. A skin graft was obtained from the patient's abdomen and allowed to lie in a circular fashion around the area of scar excision over a polyethylene tubing of suitable size. The tubing remained two months. The patient was told how to remove the tubing at daily intervals and to reinsert it after cleansing. A minor degree of contraction occurred, but complete patency was obtained. The ear canal has remained patent for the past two years. The patient's hearing was normal following the procedure, whereas previously he had a severe conduction-type loss, as would be expected.

SUMMARY

Atresia of the external ear canal may be congenital or acquired. The term congenital atresia is used to indicate closure of the ear canal together with the associated drum membrane defect and abnormalities of the middle ear structures. Formerly neglected, these patients should be treated early be-

cause of the possible tardy intellectual development from coexistent speech defect.

Previously, misunderstanding of the embryonal development of the ear resulted in poor surgical results. Only in the past eight years has the literature offered reports of consistently good results.

An understanding of the development of the ear is essential. First, the inner ear develops from ectoderm; next, the middle and the external ear develop from the gill structures. Anomalies of either portion of the ear may occur without any associated defects of the other parts of the ear. The usual finding in congenital atresia is complete bony closure of the ear with a bony eardrum and a deformed malleus and incus. The auricle is often deformed.

Acquired atresia is caused by soft-tissue or bony disease from inflammation, trauma, or neoplasm.

Surgery is primarily directed toward improvement of the conduction-type hearing loss after being certain of normal cochlear function and pneumatization of the mastoid process. The best age for correction of congenital atresia is from 2 to 4 years.

Endaural surgery of the involved ear is best, entering the antrum with removal of the deformed ossicles and bony drum if fusion of the ossicles to the drum is found. Mobility of the stapes should be determined. A split-thickness skin graft is allowed to lie against the bony eardrum, if it is allowed to remain, or against the stapes. If the stapes is fixed, fenestration may be considered.

A method using a hollow polyethylene tube with wrapping of the skin graft around the tube to fit snugly into the newly made canal is described. This insures a successful take of the skin graft, good drainage, and prevention of subsequent stenosis. Cases are presented.

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Use of the Electrolarynx in Speech Rehabilitation

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A study of several papers on the subject of the psychological aspects of laryngectomy shows a unanimity of opinion among the authors concerning the importance of the psychic trauma incident to laryngectomy.

Figi and Devine [1] and Greene [2] agree that the psychic trauma is easily as serious as the physical trauma itself and should never be underestimated in dealing with the patient after the operation. Pitkin [3] regards it as one of the most serious problems in the management of patients with carcinoma of the larynx.

Reference is made herein to the relatively high percentage of patients who, because of either physical or psychological reasons, do not attain the ability to produce adequate and acceptable esophageal speech. This significant number should elicit the interest of those dealing with the laryngectomy patient—the surgeon and the speech teacher.

Levin [4] suggests several physical and psychological reasons for failure to learn esophageal speech, such as

(1) stenosis of the esophagus after extensive removal of a widespread malignant lesion; (2) resection of the cervical portion of the esophagus; (3) multiple handicaps; for example, laryngectomy plus deafness; (4) suspected recurrence, metastasis, or multiple lesions; for example, a bronchial carcinoma in addition to the laryngeal lesion, and (5) senility or other feebleness.

McCall * reviewed 149 laryngectomy patients, reporting on 126, of whom 67 "developed a good voice" and 13 "were able to learn the act." Forty-six did not learn, and twenty-three were not reported on. Pitkin [3] reported on 61 cases, of which at least 21 did not learn to speak. Thirty-five "returned to their work or duties," and three did not answer the questionnaire.

From these and other figures of comparable ratio between those patients who were able to master esophageal speech and those who were not, it appears that approximately one-third of all patients do not learn to speak. From observation of some patients who consider themselves able esophageal speakers, the proficiency of the remaining two-thirds might be questioned.

This relatively large number of patients unable to attain adequate speech by the esophageal method, and the depression resulting from resignation to absolute silence and resort to pad-and-pencil communication, suggests the alternate choice of the use of the electrolarynx. This is an electronic instrument, basically a cylinder 4 in. in length and 1 in. in diameter, powered by a small battery. Its head, when held against the throat, produces vibrations which emerge from the mouth as sound. The patient's tongue, lips, and cheeks form the words as in normal speech. The voice produced with this instrument is monotone, but the speech is intelligible. Ambient noise from the instrument is annoying and should be lessened with adequate engineering effort. Results when it is used over the telephone are very satisfactory, for the reason that the ambient noise is not transmitted. Volume is adequate under all circumstances.

Levin [4] suggests that use of the electrolarynx is indicated when the patient cannot

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* McCall, J., in discussion on Hoople.[5]

master esophageal speech for some special reason. He states:

> Before a course of [speech] training is initiated, the patient must be examined to determine whether he is a suitable candidate. In the preliminary examination it must be established by fluoroscopy and radiography whether the hypopharyngeal pocket is ample and the upper esophageal sphincter functionally normal. At the same time the status of the respiratory function and the cardiovascular system is ascertained. The nose and throat are examined, and the hypopharynx and the esophageal mouth are inspected with a laryngeal mirror or, if necessary, an endoscope. A search is made for metastases, and the general health of the patient is considered. Hearing must be adequate.

Pitkin [3] says that, if, after an adequate trial, it becomes apparent the patient will not learn esophageal speech, it is perhaps better that the surgeon recommend the abandonment of the effort to improve the morale of the patient and that of his family. He states that in certain cases in which economic necessity demands an early return to work, mechanical larynx instruments may be helpful. In the case of some patients who are learning or have learned esophageal speech, "these instruments may serve as an aid at times of stress or when the patient has a cold."

The psychological importance of an early return to communicative ability should be considered. Speech, being a most important social function, should be restored to the patient as soon as possible. The psychological effect of enforced silence during a protracted learning period for esophageal speech is the building of resentments and frustration that tend to make the patient uncooperative. It is perhaps better to offer him the help of the electrolarynx, with which he can at least satisfy his all-important sense of speech.

With regained ability to communicate orally, the patient is enabled to project himself and divert from the repressions and frustrations resulting from enforced silence. His morale is appreciably helped, and his family and associates are put at their ease. Telephone use is made available to him. The performance of many acts of gregariousness is made possible, with the salubrious effect serving to divert the patient from psychoneurotic tendencies.

Most patients are able to produce intelligible speech with the electrolarynx after practice of a few minutes to a few hours. Esophageal speech usually requires months of practice. There is no physical exertion required in the use of the instrument. Demonstration of the electrolarynx by a well-adjusted user-patient usually helps the morale of the patient and makes acceptable to him the use of the device.

The surgeon, rather than the speech teacher, is better able to decide which patient cannot learn to speak by the esophageal method. Objectivity by the speech teacher is made difficult (if not impossible) by the very nature of his work, which demands extreme enthusiasm for the esophageal method. Furthermore, the patient who has failed in his efforts to attain adequate esophageal speech may absent himself from speech classes, whereas he will continue to visit his surgeon for periodic check-up and guidance.

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Hypometabolism in Relation to Ear, Nose, and Throat Disorders

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In ear, nose, and throat practice, as in medicine in general, modern concepts of endocrinology have diverted attention from its older knowledge. These revised concepts often have totally disregarded the significance of other pertinent factors, especially those related to the problem of metabolism. The situation, in this connection, is precisely expressed by Simpson,[1] in reporting on some recent advances in endocrinology:

> We have perhaps been rather dazzled by the influence of new hormones on the symptomatology of disease processes, and it might be argued by the pragmatic physician, or humanitarian, that successful therapy is the end-object of all medical research, but this conception certainly cannot replace the need for a deeper understanding of the causation of disease and its course.

The present study was undertaken because the problems entailed have been insufficiently dealt with in modern medical practice. "It would be a great mistake," as Simpson emphasized, "to ignore the importance of clinical observations in the light of a newer knowledge."

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TERMINOLOGY—DEFINITIONS

The main object of this paper is the consideration of the subclinical form of hypothyroidism, so-called nonmyxedematous hypothyroidism, as an indication of metabolic changes which directly or indirectly account for a number of ear, nose, and throat disorders. Although clear-cut signs and symptoms render the diagnosis of hypometabolism no difficult problem, it is the exception rather than the rule for the obvious situation to be present. No argument is required to support the view that total glandular dysfunction frequently is responsible for various symptoms and, at times, definite entities. There exists, however, unwarranted doubt concerning the role played by subclinical glandular dysfunction.

The term hypothyroidism, unless qualified, designates that state of the body in which certain symptoms and signs are clinically recognizable.[2] If these are pronounced, according to Werner,[3] an advanced state of hypothyroidism, or myxedema, exists. In its milder form, hypothyroidism is regarded as a nonmyxedematous condition with basal metabolic rates approximating 20% below normal. Although most textbooks divide hypothyroidism into a few classical types, such as childhood (cretinism) and adult myxedema, Werner[3] contends that mild degrees of hypothyroidism may and do exist which do not come within the above classification.

The question arises whether the expression hypometabolism is more appropriate than subclinical hypothyroidism or nonmyxedematous hypothyroidism. In Wil-

liams'[2] opinion, shared by me, one of the commonest misuses of the diagnosis of hypothyroidism is the application of it to patients who have hypometabolism but not hypothyroidism. Hypometabolism probably is intended to suggest a general "slowing-down" of body functions, without obvious signs or positive tests of typical hypothyroidism.

Hypometabolism and hypothyroidism without myxedema are used synonymously by Barr.[4] A lowered basal metabolic rate occurs in several diseases without any clinical signs or symptoms, according to Mason.[5] In his opinion such a state should be termed secondary hypothyroidism.

An analogy can be drawn from the discussion by Reiss and Haigh[6] on various forms of hypothyroidism in mental disorders. These writers, like so many others, stress that the clinical diagnosis of hypothyroidism is frequently overlooked. "Between the clinically obvious hypothyroid state and the normal there is a kind of no-man's land in which the true or effective level of thyroid activity is not easily decided." Even with the aid of present laboratory methods, Reiss and Haigh experienced difficulty in determining the significance of the intermediate values.

It is logical to assume that a similar situation obtains in ear, nose, and throat disorders in which an endocrine relationship is suspected and in which test values, i. e., values below the accepted standard, reveal a subclinical condition. The problem thus resolves itself into one of interpretation of values when these are considered in relation to other laboratory results, the history, and the clinical signs and symptoms. The potential influence of subclinical endocrine states on diseases in general remains a problem for further research.

LABORATORY AIDS IN DIAGNOSIS

There are several laboratory tests which prove helpful in the diagnosis of suspected endocrine diseases. Escamilla[7] pointed out, however, that laboratory findings are for the most part only aids or adjuncts, not only in diagnosis but also in following the effects of treatment in endocrine disease. This writer emphasized:

> They do not replace or transcend a proper and complete anamnesis and physical examination of the patient. . . . If the laboratory results indicate conclusions at variance with clinical facts it is prudent to place more credence in the clinical observations, and either repeat the tests (perhaps in another laboratory) or if necessary repeat or re-evaluate entire experiments.

The test more commonly employed in the past, and even at present, yields the determination of the basal metabolic rate. It remains the ideal method for most practical purposes, and while it is subject to inaccuracies from various influences, correctly and carefully performed, it serves a highly useful purpose.

More recently, use has been made of the blood protein-bound iodine test. This is a delicate test with definite advantages under certain circumstances over the basal metabolism test. In hypothyroidism, the result may be low or low normal. The extremes recognized are 3.5γ to 8γ/100 cc.

The level of plasma cholesterol often is revealing. In hypothyroidism the level is increased (300%-1000%). There is no unanimity of opinion concerning the normal range, which varies from 150γ to 190γ/100 cc. to a range between 100γ and 300γ/100 cc.

Mention is made also of the tracer dose of radioiodine, but, according to Seed,[8] it is not of much use in the diagnosis of hypothyroidism. Seed holds that the BMR as a diagnostic test is equal to or superior to the tracer dose of radioiodine. "But paradoxically, when clinically you are really in need of a diagnostic test in the borderline case, the tracer dose is more useful than the metabolic rate."

To these laboratory procedures Chinsky[9] has added the use of the McWhirter-Freibrun metabolic calculator. "The latter is a device that utilizes easily available information such as body weight, blood pressure, and pulse rate, from which basal metabolic rate is determined by a standard formula."

Evaluation and interpretation of these respective tests must be left to the judgment of the attending physician. Suffice, as previously stated, that for practical purposes and despite its deficiencies, the basal metabolism test remains the test of choice. If the results do not correspond with the symptomatology, other tests can and should be employed for purposes of verification.

CLINICAL CONSIDERATIONS

In a previous paper [10] dealing with thyroid dysfunction in relation to rhinologic disease, I emphasized the importance and value of a detailed history. Although the general impression obtained from intranasal examination is suggestive, the history often proves more revealing. Both the history and the findings on examination should be supported by results of laboratory tests for a conclusive diagnosis. Hypometabolism may be associated with the following processes:

Nasal Mucosa

Changes in the nasal mucosa as indicated by thyroid deficiency may be of two types, in the opinion of Proetz,[11] viz., (*a*) red, dry, irritated, "chapped," or desquamative; (*b*) pale and boggy. In a group of 50 patients suspected for the first time as a result of nasal examination alone the metabolic rate was found by Proetz to be 0 to —10% in 9 patients, —11% to —20% in 18 patients, —21% to —30% in 8 patients, and —31% to —40% in 4 patients. According to Proetz, awareness of thyroid insufficiency depends at present chiefly upon the awareness of the rhinologist of its existence. "Variable and vague though the symptomatology may be, the diagnosis is simple. A low basal metabolic rate is presumptive evidence; a therapeutic test is conclusive." In nasal conditions in which allergy is a factor, correction of any existing metabolic errors or endocrine dysfunction usually enhances the effect of allergic therapy.

Vasomotor Rhinitis

In vasomotor rhinitis, the diagnosis of endocrinopathy should be based mainly on the symptomatology. In 1927, Novak [12] associated hyperesthetic rhinitis with myxedema, but this undoubtedly represented an extreme viewpoint. It has been suggested that endocrine dysfunction may be one of the factors producing a sympathetic-parasympathetic imbalance and that this may be responsible for the nasal condition. This diversity of views is difficult to reconcile, though in classifying vasomotor rhinitis an endocrinopathic type must be recognized. Whether an endocrinopathy is the direct cause or merely an indirect factor in altering the body metabolism is conjectural. In Semenov's [14] opinion vasomotor rhinitis merely means that there is an erection of the erectile tissues. In a recent study conducted by me [15] on 52 patients with vasomotor rhinitis, 38 yielded a BMR on the minus side. In 18 of these 38 patients, the PBI determinations were 5γ/100 cc. or under.

Postnasal Discharge

According to Proetz,[16] among the endocrine disturbances, thyroid deficiency is by far the most frequent cause of postnasal discharge. "Changes in the hair and skin find a parallel in the nasal mucosa which is boggy and covered with viscous mucus." Richards,[17] in a comprehensive review of the subject of postnasal discharge, stated:

> The influence of the ductless glands on our whole sympathetic nervous system is so great that to associate nasal hypersecretion and changes in the constituents of nasal mucus with increased or diminished activity of the thyroid gland, the adrenal or the pituitary, is a very natural and often justifiable temptation however difficult of scientific proof. Changes in nasal secretion during pregnancy or the menopause have often been observed, and frequently offer a welcome explanation for the patient's complaint.

Deafness and Tinnitus

Hill [18] called attention to the fact that of the factors producing tubal obstruction endocrine conditions must be included. Laskiewicz [19] described the changes in the upper respiratory organs and the ear in the presence of thyroid dysfunction. As concerns the ear, he noted that the following symptoms may accrue: catarrhal affections of the

middle ear, tubal obstruction, tinnitus with marked hearing disturbances frequently accompanied by giddiness. These symptoms Laskiewicz found were refractory to local therapy but frequently reacted favorably to thyroid administration. McMahon [20] expressed the opinion that tinnitus may occur in hypothyroidism, with or without an impairment of hearing. This may be explainable, according to McMahon, on the basis of the lowered blood pressure and the atonic state of the large vessels and tissues adjacent to the middle ear and the Eustachian tube, or it may be the result of an albuminoid distention of the cochlear cells.

Lymphoid Hyperplasia of the Pharynx

It is now generally conceded that of the several factors influencing the growth of lymphoid tissue the endocrine secretions must be duly considered as of significance. Dalton [21] found that lymphoid hyperplasia was influenced by hyperthyroidism and by hypothyroidism, though the relationship of the latter to lymphoid hyperplasia was more pronounced than in hyperactivity of the thyroid gland. In a review of his office-case records, Dalton observed that 19.2% of the patients had thyroid dysfunctions. Lymphoid hyperplasia was present to a marked degree in 9.67%, to a moderate degree in 32.88%, and to a slight degree or not at all in 57.35%.

As concerns the factors influencing the growth of lymphoid tissue, White [22] stated:

> The active secretion of the thyroid, controlled by the thyrotrophic hormone of the adenohypophysis, may acutely cause reduction of lymphoid tissue size as a result of stimulation of adrenotrophin secretion. Chronically, however, thyroid secretion appears to stimulate the growth of lymphoid tissue, since enlarged lymphoid structures have been observed in experimental thyroid feeding and in clinical hyperthyroidism.

White emphasized that a wide variety of endocrine influences may alter the size and structure of lymphoid tissues. In this connection, he remarked:

> The important role which these endocrine secretions play in the normal physiology of the organism, and the wide variety of factors which influences the concentration of these circulating hormones, all point to intimate relationships between endocrine glands and the size of lymphoid tissue.

Headache

In some rhinologic diseases, headache is frequently a complaint. Reports by Bryant,[23] Seward,[24] Austin,[25] and Fox [26] suggest a possible endocrine relationship to headaches. Fox [26] described a type of nasal headache which develops in the course of vasomotor rhinitis with meager objective physical findings. Characteristically, these patients have a low BMR but no signs of myxedema. In Fox's experience, the favorable response to thyroid medication was noteworthy.

Ryan,[27] Friedman,[28] Moench,[29] and others have emphasized that dysfunction of the endocrine glands must be suspected as a likely cause of headaches whose etiology at first appears obscure. In some instances, as pointed out by Fox, the headache symptom may be associated with some mild form of nasal pathology.

Therapeutic Considerations

Of special interest in hypometabolic states is the problem of management. It is generally conceded that thyroid medication is effective in controlling the symptoms. There are, however, differences of opinion concerning the desirability of administering thyroid to patients with hypometabolism. Thorn [30] is opposed to such treatment because ultimately, with the increasing dosage usually required, "the total dose of thyroid exceeds that which the gland normally puts out, and persistent signs of hyperthyroidism become evident."

Williams,[2] likewise, believes that whereas patients with hypometabolism show immediate improvement when given desiccated thyroid they soon become refractory. "With an increase in the dosage they again observe a response. However, after several years of a progressive increase in dosage such a patient may state that he notices no significant difference in his symptoms whether he is or is not taking thyroid."

In disagreement with these negative views on thyroid medication, McLaurin [81] wrote:

> Throughout the literature on hypothyroidism the necessity of the patient to continue taking thyroid is stressed. This is not true in the cases of milder hypometabolism. After the patient is free of symptoms, the dose can be decreased gradually and the therapy finally stopped, when the metabolism has been brought to normal limits.

Of the two schools of thought I am inclined to lean toward McLaurin's,[81] which holds that in the nonmyxedematous form of hypothyroidism (hypometabolism) administration of thyroid can eventually be discontinued without deleterious influence. Since resort is had to a maintenance dose of the drug after a tapering-off process, this dosage is usually minimal. The important consideration during the entire procedure is to search for and correct factors known to alter the metabolic state. Mental strain, psychological factors, nutritional indiscretions, and intercurrent illnesses may lower the metabolism appreciably. A low blood pressure, a slow pulse, and generalized fatigue are common symptoms. When these are corrected, the metabolism can be brought up to the normal range without the need for stimulating the hormonal system. Unless efforts are directed in accordance with this plan, it should be obvious that thyroid medication will serve only a palliative purpose.

It has been maintained in some quarters that a depression of less than 20% below the average normal does not necessarily indicate underfunction of the thyroid gland. This has not been the more general experience based on therapeutic tests. For example, in patients with basal rates of —10 results from thyroid therapy have been as dramatic as in those whose basal rates were —25 or —30.

REGULATION OF THYROID DOSAGE

Though the BMR serves as a guide to dosage, and should be utilized to control it, the patient's tolerance for the drug must also be considered. It is suggested that in adults the trial dose of thyroid extract be restricted to ½ grain (30 mg.) daily (or its equivalent, depending on the product employed). From this base, the dose should be stepped up in relation to the individual patient's response.

It must be borne in mind that U. S. P. animal products of thyroid vary in strength, and, therefore, regulation of dosage often becomes a difficult problem. This may be overcome by use of the product known as sodium levothyroxine (Synthroid). It contains levothyroxine, the active principle of the thyroid gland, as the monosodium salt, prepared synthetically in pure crystalline form. The product is marketed in scored tablets containing 0.05, 0.1, or 0.2 mg. of sodium levothyroxine. For purposes of comparison, the activity of a 0.1 mg. tablet of sodium levothyroxine is equivalent to that of approximately 1 grain (60 mg.) of thyroid U. S. P.

Patients Classified According to the Disorder Present

Disorder	Patients			
	Total No.	Showed Minus BMR Readings	Improved Under Treatment	
			No.	%
Vasomotor rhinitis	52	38	26	68
Postnasal discharge	28	16	11	68
Deafness and/or tinnitus	40	26	18	69
Lymphoid hyperplasia of pharynx	51	27	14	51
Headache	31	19	11	57

Studies [38] have been conducted to evaluate sodium levothyroxine administered orally for hypothyroidism. The results have been highly favorable. The dosage must be governed by determination of the systemic condition, the various tests commonly employed, and the efficiency of the patient.

DETAILS OF STUDY

The present study included a total of 202 patients who were classified into categories as listed in the Table. The Table shows also the number in each category who gave minus BMR readings. Only the latter were subjected to thyroid medication and/or correction of errors of metabolism. The Table reveals also the number of these patients who improved under indicated therapy.

The selection of patients for testing was made merely on the basis of suspicion, because of suggestive clinical symptoms, or because the cause of the complaint was obscure. It will be noted that of the total of 202 patients (without regard for the diagnosis) 126 yielded minus BMR readings.

GENERAL COMMENT AND CONCLUSIONS

From the facts presented, it should be clear that in certain otolaryngologic disorders even a mild hypometabolic state may be the influencing factor. Because the symptoms and signs are not clear-cut and, therefore, cannot be easily recognized, the likelihood of endocrine dysfunction is rarely considered. Experience has demonstrated that many clinicians regard subclinical hypothyroidism as of minor significance. It should be stressed that some degree of thyroid dysfunction may be present despite the fact that measured thyroid activity is normal.[6] Results of therapeutic tests often serve as convincing evidence of functional irregularities.

Finally, it should be stated that the futility of topical therapeutic methods in many ear, nose, and throat disorders probably can be accounted for by lack of appreciation of the influence of endocrine dysfunction on body metabolism.

SUMMARY

In ear, nose, and throat practice modern concepts concerning hormones have diverted attention from the older knowledge of endocrinology.

Some confusion exists concerning the terminology employed to designate the subclinical form of hypothyroidism—hypometabolism—in which classical myxedematous signs and symptoms fail to manifest themselves.

Between the clinically obvious hypothyroid state and the normal "there is a kind of no-man's land" in which the true or effective level of thyroid activity is not easily decided.[6]

In persons in whom a low BMR is the only indication of hypothyroidism, clinical symptoms being absent, the dysfunction is probably a secondary process, as suggested by Mason.[5]

Laboratory tests for endocrine disease serve as aids in diagnosis and as guides in controlling therapy.

Nonmyxedematous hypothyroidism—hypometabolism—produces definite changes in the nasal mucosa, is one of the underlying causes of vasomotor rhinitis, and frequently accounts for persistent postnasal discharge and, occasionally, for refractoriness of tubal obstruction, deafness, and tinnitus. In growth of pharyngeal lymphoid tissue and in some headaches, alterations in metabolism sometimes play a prominent etiologic role.

Contrary to the general belief, patients with subclinical hypothyroidism do not become resistant to thyroid therapy; in fact, the drug can be withdrawn without disturbing effects if the causes of the hypometabolism are eliminated or corrected.

Because of the variable strength of U. S. P. products of thyroid, successful use in indicated cases has been made of sodium levothyroxine (Synthroid), containing levothyroxine, the active principle of the thyroid gland, as the monosodium salt, prepared synthetically in pure crystalline form.

A series of 202 patients was studied, 126 yielding minus BMR readings. In the aggregate, although a breakdown is given in the text, approximately 60% of the 126 patients treated had satisfactory results.

Lack of appreciation of the part played by endocrine dysfunction, more especially hypometabolism, in certain ear, nose, and throat disorders could logically explain the futility of some time-honored local procedures.

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Intracranial Complications of Chronic Mastoiditis

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Is mastoiditis and its life-threatening complications a disease of the past? Many are under this impression. It is not uncommon to be asked, "Do you still do mastoids?" "I thought that penicillin had changed all that." It is our obligation as otolaryngologists to dispel this cloud of misinformation. I wonder how many of the readers of this paper have seen mastoiditis temporarily camouflaged by the antibiotics suddenly erupt into brain abscess or meningitis. I would venture to say that most of us have seen at least one case of this sort recently, and I predict that we will see increasing numbers, with the resistant organisms multiplying daily and with the rapid increment of allergic reactions to the wonder drugs. It has been estimated that 6% of all patients are allergic to penicillin,[1] and the number is growing rapidly.

What are the complications of mastoiditis? For the most part they are secondary to chronic disease. Some of the more serious complications of mastoiditis are (1) facial nerve paralysis, (2) labyrinthitis, (3) petrositis, (4) lateral sinus thrombosis, (5) brain abscess, and (6) meningitis.

In this paper I cannot begin to cover these complications even in part. However, I will present three cases which illustrate nearly classical examples of several of these extensions of chronic mastoid disease.

REPORT OF CASES

CASE 1.—A white man, aged 39, gave a history of right otorrhea of 12 years' duration. Two weeks prior to admission he developed symptoms which he thought to be the "flu." For two to three days just before admission, he noted some pain on flexing his neck, fever, and severe chills. He continued on his job as milkman and made his morning milk run the day of admission.

On physical examination, the temperature was 101 F, pulse rate 100, respiration rate 24, and blood pressure 128/62. The patient was an alert, well-nourished white man. He appeared to be acutely ill. The right external auditory canal was filled with foul purulent drainage. The tympanic membrane was largely destroyed, and the middle ear was filled with granulation tissue. The mastoid was tender over the antrum. There was definite stiffness of the neck.

A spinal tap was performed and the fluid found to be cloudy. On microscopic examination there were 10,500 cells, 90% polymorphonuclears and 10% lymphocytes. No organisms were seen on direct smear, but numerous coliform bacilli were isolated on culture. A blood culture was sterile.

X-rays of the mastoids were reported as showing sclerosis and underdevelopment of the right mastoid but no evidence of cholesteatoma or bone destruction.

The patient was taken to the operating room and a postauricular radical mastoidectomy performed. The mastoid was found to be very sclerotic. A small antral cholesteatoma was encountered. Over the midportion of the sinus plate a definite erosion was found with an exposure of the lateral sinus. The lateral sinus was exposed throughout its mastoid course and opened with a longitudinal incision and a large granular thrombus removed. This was followed by free bleeding from each end of the sinus. The sinus was packed with sulfathiazole packing and the usual dressing applied.

Postoperatively the patient did well. His only complaint was headache, which persisted for two weeks and gradually subsided. Large doses of the antibiotics were continued during his convalescence. He was discharged on his 14th hospital day and followed as an outpatient.

CASE 2.—A white man, aged 35, gave a history of otorrhea of 15 years' duration. Approximately 10 years prior to his admission, he developed severe pain in his right ear and apparently a facial nerve paralysis. This cleared up, and he was well except

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for foul otorrhea until two weeks prior to his hospitalization, at which time the drainage ceased and the right temporal headache was noted. This became gradually worse, and his local physician gave him large doses of penicillin and erythromycin. Two days prior to admission severe chills and fever were noted. Upon admission, the temperature was 99.4 F, pulse rate 85, respiration rate 20, and blood pressure 130/72. The patient was lethargic. The right external auditory canal was filled with a purulent foul discharge. The tympanic membrane was destroyed and the middle ear filled with granulation tissue. The right mastoid was tender over the antrum. The optic discs showed very early choking, with distention of the veins. The neurological examination was negative except for nuchal rigidity.

X-rays of the mastoids revealed a very sclerotic mastoid, with an area of bony erosion over the lateral sinus plate.

He was taken to the operating room and a ventriculogram attempted, without success. This was followed by an endaural mastoidectomy with local anesthesia. When the area of the lateral sinus was reached, there was a release of a large amount of purulent material. There was a very definite erosion of the sinus plate. The dura of the posterior fossa was widely exposed and found to be thickened, tense, and covered with granulation tissue. The dura overlying the tegmen tympani was also widely exposed and appeared normal. It was the very definite opinion of the neurosurgeon that nothing further should be done at that time. In retrospect, I believe this was a serious error.

Postoperatively, the patient did quite well until the sixth day, when he became lethargic, developed marked papilledema and definite signs of a temporal lobe abscess. Another ventriculogram was made, which revealed a shift of the ventricular system to the left. A craniotomy followed immediately, and an abscess was encountered 1.5 cm. from the surface of the temporal lobe, just above the ear. Catheter drainage was instituted.

Cultures taken at the time of surgery revealed Gram-positive cocci and Gram-negative rods which were resistant to all the antibiotics except chloramphenicol (Chloromycetin) and nitrofurantoin (Furadantin).

During the next few weeks the mastoid cavity was treated with local hygiene and insufflations of iodine-boric acid powder. The catheter in the abscess was irrigated with a solution of chloramphenicol. Large doses of chloramphenicol were given throughout this period systemically. The patient responded slowly and was discharged on his 36th hospital day.

Three days later the patient was readmitted in a comatose condition, with projective vomiting, marked papilledema, and a left central seventh nerve paresis. The craniotomy incision was reopened, and a total excision of a right posterior temporal lobe abscess carried out. The patient seemed to improve for a short time but died five days after his second admission.

Necropsy revealed the brain to be tense. As the brain was removed a large quantity of greenish exudate was found along the tentorium, and upon incision of the tentorium purulent material was found within the lateral sinus. A large abscess was present in the right cerebellar hemisphere and on section several smaller abscesses within the cerebellum, one being very close to the midline and involving a portion of the brain stem.

CASE 3.—A white man, aged 30, was admitted to the hospital in a disoriented semicomatose condition. There was a heavy odor of alcohol on his breath. A rather sketchy history was obtained from a sister. Apparently this man was well until four days prior to his admission, when he complained of headache and right otalgia. On the day of admission, chills and fever were noted and he became disoriented. The patient had engaged in a bout of heavy drinking for several days prior to his admission. A tentative diagnosis of delirium tremens was made and chronic otitis media, right, with possible mastoiditis. The patient was placed in a locked observation ward and was started on antibiotic therapy after culture material was obtained.

Clinical examination shortly after admission revealed the temperature to be 100 F; pulse rate 100, and blood pressure 140/80. The right external canal contained a profuse amount of foul drainage. The right mastoid was tender over the antrum and over the tip, and the entire right temporal region was tender to palpation. There was some redness and swelling over the right mastoid. The tympanic membrane was largely destroyed, and the middle ear was filled with granulation tissue and a moderate-sized polyp. Except for the ear pathology there were no significant findings upon physical examination.

The day following admission it was noted that the patient had marked nuchal rigidity. A lumbar puncture was performed, and the fluid was found to be grossly cloudy. The pressure was 400 mm. of water.

A neurosurgical opinion was obtained. There were no gross visual field disturbances. There was no nystagmus. There were no focal signs of brain abscess.

The following laboratory studies were obtained: X-rays of the skull revealed a normal skull. Both mastoids were sclerotic, and an area of possible erosion of the lateral sinus plate was noted on the right.

Bacteriologic studies of the discharge from the right ear revealed a Proteus organism which was resistant to all of the antibiotics except chloramphenicol, chlortetracycline (Aureomycin), and oxy-

tetracycline (Terramycin). The white blood cell count was reported as 12,150, 78% of which were polymorphonuclears. The spinal fluid revealed 80% of the cells to be polymorphonuclears.

The patient was taken to surgery, and with use of endotracheal anesthesia an endaural incision was made. In the region of the spine of Henle there was noted a very definite perforation of the cortex. The mastoid cells were gradually exenterated, and in the region of the antrum a collection of foul pus under pressure was found. The purulent material exuded from the region of the antrum and continued to pour forth until approximately 2 to 3 oz. of pus was obtained by aspiration. The mastoid antrum was gradually exposed and appeared to be filled by what was first thought to be a huge cholesteatoma. With further exposure, however, it was obvious that this represented a herniated or prolapsed dura which completely filled the attic and antral regions. The dura was tremendously thickened and covered by a whitish plastic exudate. The lateral sinus was exposed and found to be normal in appearance and upon palpation. The entire tegmen tympani was destroyed, allowing the herniation of the dura of the middle fossa. The facial nerve was found to be exposed pathologically in the middle ear. The radical mastoidectomy was completed, and a rather wide incision was made through the dura. This incision was extended so that by palpation it was determined that almost the entire temporal lobe was destroyed. A No. 10 rubber catheter was inserted into the abscess cavity and sutured into position, and the mastoid cavity was lightly packed with iodoform gauze. The usual dressing was applied.

Postoperatively the patient did quite well. He was completely rational several hours after returning from the operating room and offered little in the way of complaints during the postoperative period. His temperature returned to normal on his second postoperative day. The catheter within the abscessed cavity was irrigated with a chloramphenicol solution daily and gradually shortened. The catheter was finally removed on the 10th postoperative day. The mastoid cavity was treated with "masterful neglect." The patient was placed on systemic chloramphenicol and sulfisoxazole (Gantrisin), first by injection and later by mouth. The patient was discharged on his 21st hospital day and has been followed for three months. He was clinically well when last seen.

COMMENT

There are a number of important points which these three cases illustrate, and I should like to enumerate on two of these in a rather cursory manner for emphasis.

1. Brain abscess. Chronic mastoiditis is the commonest source of brain abscess. Acute otitis media and paranasal sinusitis account for most of the other cases, while a distant focus of infection is much less common. The infection reaches the central nervous system either by direct extension or by the hematogenous route. Stuart and his group[2] recently called attention to an apparent increasing number of cases which act like early brain abscess but resolve without progressing to actual abscess formation. They feel these cases are due to a thrombophlebitis of the cerebral veins and suggest the increase may well be due to the antibiotics which arrest the process just short of brain abscess.

What are the signs and symptoms of brain abscess? Unfortunately, the onset may be very insidious and the objective signs meager or absent. It is truly amazing how much destruction of brain tissue may be present with practically no localizing signs. The history of chronic otorrhea is highly significant. The physical findings of chronic otitis media and mastoiditis are familiar to all of us. In the presence of chronic otorrhea we must be suspicious of constant severe headache, somnolence, and low-grade fever. The usual course is that of chills and fever, malaise, headaches, vomiting, all of which may be followed by a latent period of a week to several months. Again it is important that the antibiotics may mask the picture and make the diagnosis even more difficult. During the latent period there may be no signs or symptoms. However, the eventual termination is a sudden flare-up of the symptoms and very often death in a few days or in several weeks. Terminally the abscess often ruptures into the ventricle, subarachnoid space, or into the lateral sinus.

Neurologically, the localizing signs, if present, depend on the location of the lesion. In a classical temporal lobe abscess, the findings are (1) sensory aphasia, if the dominant lobe is involved; (2) homonomous hemianopsia; (3) signs of increased intracranial pressure. Papilledema is usually late. Hearing loss is of little value in establishing a diagnosis. The entire temporal lobe may

be removed, with a resultant loss of approximately 5 db.* The only single central lesion which can produce significant deafness is a lesion in the midbrain close to the pineal gland.

With a cerebellar abscess the signs may be even more meager and often the diagnosis is evident only terminally. The classical signs are asynergy, nystagmus, past-pointing, slurring of speech, positive Rhomberg, and marked interference of gait. Terminally there may be involvement of other cranial nerves.

2. Lateral sinus thrombosis. Lateral sinus thrombosis is a result of direct extension of mastoid infection or may be secondary to a thrombophlebitis of the small veins of the mucous membrane of the middle ear. These veins communicate directly with the lateral sinus. Often there is a perisinus abscess which is followed by phlebitis and the thrombosis. The thrombus may propagate in either direction. It may be sterile or infected. The main danger of an infected thrombus is the showering of the blood stream with infected emboli.

How may we recognize lateral sinus thrombosis? The onset is usually marked by chills and fever. Headache is a consistently constant complaint. Brunner states that many of these cases with full-blown sinus thrombosis will walk into the office under their own power. However, they do look extremely ill. With progression nausea, vomiting, shock, convulsions, and stupor ensue.

The spinal tap with the Queckenstedt test or a modification (Tobey-Ayer) of Crowe's test will greatly aid in the diagnosis. However, because of anatomical variations in the venous sinuses these tests are not completely reliable.

Finally, a few words concerning therapy:

First, the antibiotics are, of course, invaluable in the treatment of otitis media and mastoiditis. Cultures with sensitivity tests are of great value in the selection of the drug or combination of drugs. A few years ago, Crowe [2] reported that 50% of the cases of lateral sinus thrombosis were due to a hemolytic Streptococcus. The flora of these ears has changed. Since the widespread application of the wonder drugs, we are encountering resistant strains and organisms formerly considered as saprophytic are emerging as pathogens.

Second, the value of the anticoagulants has not been ascertained, but certainly, in view of their application for thrombophlebitis in other fields, they would seem a valuable adjunct to therapy. Even with the antibiotics and anticoagulants ligation of the internal jugular vein is necessary in some cases. Whether it should be a routine procedure is debatable.

Third, when we are faced with a definite brain abscess which has involved the brain by a direct extension from the mastoid, I feel that it is our obligation to attempt decompression and drainage through the mastoid cavity. Some of the neurosurgeons will disagree on this point.

Lastly, the best possible treatment is prevention. Most of these ears give adequate warning of impending disaster. Most of them have been treated from time to time by competent otolaryngologists. I firmly believe that we must place increasing importance on the clinical examination and less emphasis on the x-ray. The absence of x-ray evidence of cholesteatoma or bone destruction should not relax our vigilance. Many of the sclerotic mastoids by roentgen examination are potentially dangerous. The answer is earlier surgical therapy. These ears must be made safe by complete and adequate mastoidectomy.

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Temporal Bone Surgery in Otitis Media and Mastoiditis

Indications, Purposes, and Aims of the Different Operative Procedures

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After a decade of general use of some of the most effective antibiotic and sulfonamide drugs, one finds that infectious disease of the middle-ear spaces and the mastoid portion of the temporal bone still accounts for the majority of all cases of deafness encountered in children and young adults. Admittedly, the number and severity of dangerous complications of middle-ear infections are much less than in the preantibiotic era; nor is the amount of chronicity and deafness as great. Yet, the considerable number of cases in which pathological changes of a chronic nature affect the sound-conducting apparatus makes it apparent that, apart from the sulfonamides and antibiotics, there exists need for improvement in the diagnosis and treatment of these conditions.

Along with the advances in medical treatment of all infectious diseases, including those affecting the ear, has come the refinement of surgical techniques developed primarily to perfect the fenestration operation for the restoration of hearing in many cases of otosclerosis. Even if the fenestration operation had failed to attain its present high degree of success, there would remain the very important byproduct, so to speak, of its accomplishment, namely, the perfection of the techniques of temporal bone surgery for conditions other than otosclerosis.

We feel there is need for an up-to-date understanding of the indications for temporal bone surgery in infectious ear diseases, as they have been modified by the antibiotics. There is, to be sure, a place for "conservative" treatment of any disease. However, using surgery as "a last resort" is often less than fair to the patient. Where the ear is concerned and the aim is to preserve hearing, timeliness of surgery is, in our view, the single most important factor influencing the outcome of a given procedure.

Anatomically the temporal bone is subject to only a few well-recognized deviations from the normal. But the response to the pathology of infections, and indirectly to treatment, is variable. Therefore, each case must be individualized in practice. If this is remembered when speaking in a general way of the purposes and aims of the various surgical procedures, it will help to prevent this presentation from becoming unwieldy through the repetition of case histories.

MYRINGOTOMY

Not every case of acute otitis media requires incision of the tympanic membrane. If the ear is seen within a few hours of the onset of pain, even if the drum is hyperemic and slightly bulging, it is usually safe in the absence of general signs to defer myringotomy. If improvement does not follow the administration of the antibiotic and chemotherapeutic agents in at least 24 hours, myringotomy should be performed, especially if there is (1) tympanic inflammation, (2) fever, (3) pain, (4) reduction of hearing,

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(5) chills, (6) cervical rigidity, (7) postauricular tenderness. Sometimes an ear already draining may still require myringotomy. This is always true if pain recurs after the cessation of the discharge.

In infants there are several additional considerations. The tympanic membrane is normally thicker, and bulging is apt to occur later than in older children and adults. Many cases of chronicity date from infancy, and a paracentesis of the eardrum should be done on less typical indications. Where convulsions occur, not otherwise very apparent as to cause, bilateral myringotomy should be done on the very slightest changes in the tympanic membrane.

Wherever there is bulging in Shrapnell's membrane, although the remainder of the drum may be normal or nearly so, myringotomy is indicated. Bulging here indicates that the folds or pouches of Tröltsch, normally serving to fend off invasion of the epitympanum, have been trespassed upon; then, paradoxically, they help to confine infection in the epitympanum, where it is most dangerous to the hearing. Myringotomy should usually include Shrapnell's membrane and the posterior ligament of the annulus. It is then not likely to require repeating.

In serous otitis, usually the drum does not bulge; it may actually be retracted. This is especially true of the mucoid type seen more often in children in contrast with the more fluid exudate common to adult cases. Although cultures may be negative, almost always a low-grade infection is present. Fibrinous organization of the exudate may follow and a chronic adhesive process develop from this state of affairs. It is advisable in cases of serous otitis, where myringotomy is done, to follow incision of the drum with immediate catheterization of the Eustachian tube. Sometimes a ropy mucoid exudate can be pushed through the Eustachian tube and into the nasopharynx by gentle pressure with the Siegle otoscope. Frequently in these cases, diminished hearing is the only symptom aside from a blocked sensation. Fever and pain are usually absent. Inflation of the Eustachian tubes, via a catheter or Politzerization, may have to be repeated a number of times before the hearing returns to normal. Audiograms should precede and follow each inflation. In the acute suppurative cases the same procedure should follow myringotomy after a reasonable time interval in case hearing tests do not return to normal. The middle ear must be inflated to prevent adhesions from forming around the middle-ear structures or the windows conducting sound to the inner ear.

General anesthesia should be used for myringotomy in all cases of acute suppurative otitis media, and in all cases of any type in infants and children. Premedication should be given and the usual precautions taken to insure an empty stomach. An ether rausch is satisfactory for infants and very small children, if everything is in readiness to perform the myringotomy at the right moment. Local anesthesia is usually sufficient in those cases of serous otitis of adults which require opening of the drum.

Cases of bullous myringitis, where the infecting organism is usually Hemophilus influenzae, usually show no middle-ear involvement. Paracentesis of the eardrum is not desirable here, for fear of introducing infection into the middle ear. Anesthetic ear drops containing glycerin will usually ease the pain.

Myringotomy is sometimes valuable in cases of acute flare-up of chronic otitis media where an existing perforation of the drum may be too small to provide exit of accumulated middle-ear secretions. Some otologists have recommended paracentesis at the time of simple mastoidectomy. This scarcely seems necessary where the operation provides drainage from the antrum outward.

THE SIMPLE MASTOIDECTOMY

Although the need for a simple mastoidectomy has naturally become infrequent as the incidence of complications of middle-ear disease has decreased, it is noteworthy that even before the antibiotic era skillfully treated cases of otitis media did not come to mastoidectomy in anything like the number which is suggested by exaggerated compari-

son. While formerly the primary purpose of the simple mastoidectomy was to prevent the frequently fatal complications of the extension of mastoiditis as an acute process, today in most cases the principal aim is to preserve hearing by taking the drain off the middle-ear spaces. Now one rarely sees the acute symptoms and classical signs of acute mastoiditis. But it does not necessarily follow that the pathology of the disease is therefore in a given case less devastating to the hearing. One sees a number of cases of chronic otitis media occurring in children who have lived all their days in the era of antibiotic and chemotherapeutic drugs. A few of them present the indications for a more radical mastoid operation. The symptoms of acute mastoiditis have often been masked by the antibiotics, while cellular changes, even to complete destruction, accompany a smoldering mastoid infection. These observations indicate that the simple mastoidectomy may have been put on the historical shelf and forgotten too readily. There are several reasons for this. Among them, the less frequent need for the procedure has probably resulted in some want of surgical skill that comes through practice; just as the reverse, that of overexercising a familiar skill, probably resulted formerly in the use of this operation more frequently than actual need dictated.

It is now more important than ever before to obtain precise bacteriological information before antibiotics are used. It still makes a great deal of difference whether temporal bone infection is caused by Staphylococcus or H. influenzae organisms, on the one hand, or by Type 3 Diplococcus pneumoniae or Klebsiella pneumoniae (Friedländer's bacillus), on the other hand. The latter two are quite apt to be associated with those cases of minimal symptomatology but rapid pathological changes. The phenomenon of bacteriological resistance becomes increasingly evident. Often, too, there is considerable disparity between the in vitro and in vivo responses of infecting organisms to the antibiotic agents. Fortunately, we can rely upon the roentgenologist for a great deal of help in the diagnosis of not only mastoid pathology but also structural alterations in the middle ear itself.

The use of antibiotic and chemotherapeutic agents has telescoped the entire course of acute otitic disease from the former average of five or six weeks to one week or less in the majority of cases. Thus, it is important to emphasize the time of the occurrence of certain symptoms. Postaural tenderness occurring early in the course of acute otitis media means little; this same sign appearing in the second or third week of the disease is very significant. When the hearing loss fails to improve by the end of the second week, or if having partially returned it then regresses, one feels that medical treatment may have to be supplemented. If an ear continues to discharge for three weeks and the hearing fails to return, simple mastoidectomy may be indicated. The amount of discharge, even in the absence of acute signs, is significant, more especially if when it stops pain recurs or fever increases. A suppurative ear that requires repeated paracenteses most probably needs a simple mastoidectomy. Vertigo, facial paralysis, and abducens paralysis occurring in the early days of an acute otitis indicate myringotomy and maximum antibiotic treatment; these same signs occurring in the third or fourth week of an acute episode are very significant and indicate at least simple mastoidectomy. Headache occurring any time in the course of acute otitis media is a dangerous sign. Untoward general signs occurring early in the course of acute infection usually mean involvement of the meninges or labyrinth and surrounding structures by inflammatory reaction, while these signs occurring later are more apt to be due to cellular destruction and direct infection. Very rarely the middle-ear infection will have completely resolved. Perhaps the hearing has returned to normal, yet infection persists in an isolated group of mastoid cells, and the first indication that all is not well may be the onset of meningitis or labyrinthitis. In children, the onset of vomiting and diarrhea after the apparent resolution of an acute otitis media should

alert the otologist to the possibility of mastoiditis.

Prevention of chronicity and preservation of hearing are the chief aims in all but a few complicated cases where simple mastoidectomy is indicated. However, the operation will fail to achieve its goal unless it is both complete and timely. With respect to completeness, each of the well-known cellular regions in a pneumatic mastoid must be followed out. One should go as far as the anatomy and pathology allow. Unexplored cells may defeat the purpose of this operation. As regards timeliness, in view of the changed, less obvious symptomatology of these infections, it is relatively easy to miss the time of optimum benefit from this procedure. Once the epitympanic disease becomes chronic, whether or not hearing is intact, simple mastoidectomy will nearly always fail to benefit the patient, and some more radical form of mastoidectomy will be necessary. Some of the chronic cases that one now sees give a history of previous simple mastoidectomy which was performed after an ear had been discharging for three months or longer after an acute episode. Obviously this is too late to expect much from a simple procedure. Where chronicity develops after an operation that has been timely, either cells have been left or, if some years have elapsed, further pneumatization of bone may have occurred and reinfection of the new cells.

If, after this operation, an ear continues to discharge much more than three weeks, revision of the simple mastoidectomy may be indicated.

THE MODIFIED RADICAL MASTOIDECTOMY

While the antibiotic and chemotherapeutic agents are useful in the treatment of acute conditions or in acute flare-ups of chronic infections, they are unable to restore function which is lost as a result of chronic changes in the middle-ear structures and the labyrinth. Functional loss is of two types as a rule: that involving structural anatomy in the middle-ear spaces which serve to conduct sound to the inner ear and that which involves the labyrinth itself, resulting in some degree of perceptive deafness as well. It is the aim of the modified radical mastoidectomy to prevent progressive chronicity before such functional loss occurs.

Improved techniques of temporal bone surgery have greatly enlarged the field of usefulness of the modified radical procedure. Yet, it is still a compromise operation in many respects. About 40% of the cases on which it is done will eventually require a complete radical operation. Its greatest usefulness is in those cases associated with cholesteatoma of the epitympanum, mastoid antrum, and periantral cells, where a threat to hearing is present but where the hearing is, as yet, but slightly impaired. It is also indicated in those cases of bilateral chronic otitis where the hearing in one ear is useless and that in the better ear is down to, say, 6 ft. of conversation. This is all the hearing such a patient has, and every effort must be made to salvage it. In the usual case where this procedure is indicated, the mastoid portion of the temporal bone is sclerosed. Conservative measures have been tried and failed. For best results the pars tensa of the eardrum should be intact; it is then possible after removal of epitympanic disease to seal off the middle ear, thereby obtaining conditions more favorable to good hearing.

A number of cases of chronic disease, too many, are regrettably seen after the time when a modified radical procedure would be expected to work. Hearing has fallen off, necrosis of the ossicles is present, and the cholesteatoma cavity is quite large. Since the operation depends upon maintaining the integrity of the ossicular chain, great care must be taken in performing this operation to avoid injury or dislodgment of the ossicles. But if the incus should accidentally become subluxated, it should be left in situ and not removed, for nature sometimes fills in such a breech more perfectly than one has any right to expect!

As much of a simple mastoidectomy should be done as is necessary to remove all dis-

eased cells. The antrum and epitympanum are enlarged and freed of cholesteatoma. The posterior canal wall is taken down to the tympanic ring or just proximal to it. The facial ridge is lowered to the level of the horizontal semicircular canal. The endaural incisions of Lempert allow for mobilization of the canal-wall skin to form a flap covering the epitympanum and sealing it. We feel that there is no need for grafting skin in a cholesteatoma cavity, especially if such a cavity is not very large.

In cases of dense granulations about the ossicles, a modified radical procedure is less apt to be helpful; but patience in removing these granulations from the accessible portions of the ossicles is sometimes rewarded with improved hearing. The use of magnification, either by loupe or ear microscope, greatly facilitates this phase of the work.

Sometimes it happens that a well-executed modified radical operation has failed because prior attention had not been given to infection of the sinuses or of lymphoid tissue in the nasopharynx or tubal infection itself. These intermediate steps should not be overlooked in any case of middle-ear disease, but it is especially inexcusable to find them present at the last favorable moment for preserving hearing by a modified radical procedure.

In summary, the modified radical procedure is a last attempt to preserve serviceable hearing. It is never indicated for the already complicated case where perhaps facial paralysis, labyrinthitis, or intracranial extension has occurred. One must not be reluctant, at times, to operate upon an ear producing very few symptoms; for it is apparent that if much symptomatology is present, the operation is not well indicated.

AURAL POLYPECTOMY

There is still a place for aural polypectomy in the treatment of chronic otitis where the pathology is confined more or less to the mesotympanum and hypotympanum. In these cases removal of a polyp may provide sufficient drainage from the middle ear so that, along with local medical treatment, the ear may dry up completely. Where the polyp is very small, the use of a chromic-acid bead to cauterize it is often all that is needed. Where one resorts to radical mastoidectomy in every case of middle-ear polyps, especially if the mastoid is sclerosed, one may succeed only in converting a small infected cavity into a large infected cavity. In other cases, where cholesteatoma is also present, complete radical mastoidectomy is the procedure of choice. Polyps extruding through a perforation in such a way as to block secretions and exudate in the middle ear are apt to be associated with the worst intracranial complications. One objection to snaring out a polyp at the drum is the uncertainty of the site of attachment of its base, sometimes to the stapes or to a dehiscent spot in the facial canal.

OSSICULECTOMY

Ossiculectomy, formerly a popular procedure for treating chronic suppurative otitis associated with caries of the ossicles, has now become virtually obsolete. One has the feeling it might be revived with benefit in the occasional case. It, too, is a compromise procedure, and many cases in which it is indicated would eventually come to complete radical mastoidectomy. The operation has been supplanted by the endaural modified radical surgery.

THE COMPLETE RADICAL MASTOIDECTOMY

The most frequent indications for a complete radical mastoidectomy are (1) chronic suppurative otitis media where intracranial complications threaten or have occurred, (2) where the ear to be operated on is deaf or nearly so and the other ear has good hearing, (3) where the disease is primarily epitympanic, and (4) cases of aural vertigo in chronic suppurative otitis. The operation is not done, except in the presence of dangerous complications, where serviceable hearing exists. One does not expect recovery or improvement of hearing as a result of this procedure. It should always be explained to the patient just what is expected to be accomplished by an operation of this sort. It occasionally happens in skin-grafted cases

that the graft will adhere to the head of the stapes or the margin of the oval window in such a way as to trap air beneath it and thus serve as a sort of vibrating membrane, and fairly good hearing results. Obviously this cannot happen except where a movable stapes is present, and Campbell [1] has pointed out how frequently the stapes is missing in chronic otitis with suppuration.

The aim of the complete radical procedure is to convert the mastoid, mastoid antrum, and the middle ear into one continuous cavity. Infection is thereby removed from vital areas, such as the dura, facial nerve, labyrinth, and lateral sinus. Most ears eventually become completely dry. In about 85% of the cases where this operation is indicated, the pathology is confined to the middle ear and mastoid antrum, the remainder of the mastoid being sclerosed. In the other 15% or so of the cases, the mastoid is pneumatic or largely so; then, of course, one must first do an operation resembling the simple mastoidectomy before converting it into a radical. But in the majority of cases the radical mastoidectomy should be kept as simple as possible. The procedure should be adapted to suit the pathology encountered as the operation proceeds rather than follow a set pattern decided upon in advance of the operation. For example, a number of cases will be found well suited to the type of radical mastoidectomy first described by Zaufal,[3] of Prague, in 1894. Here the posterior canal wall is taken down from the beginning of the operation, the antrum and epitympanum enlarged by a miniature Kerrison punch. This, however, is not a procedure for the occasional operator and should be done by one familiar with the conventional radical mastoidectomy. Actually this is a modification of the complete radical operation.

There is a prevalent misconception that only those cases associated with cholesteatoma are dangerous as regards the development of intracranial complications. Indeed, just as many of the complicated cases occur where cholesteatoma is not a feature of the case at all. In the absence of cholesteatoma or of threatened complications, but where the pathology is still mainly epitympanic, a different approach is required. Here the purpose of the operation may be to get a dry ear after years of disagreeable otorrhea. The salutary effect of removing such a focus of infection upon the patient's general health is often striking. These are the cases associated with granulations of the middle-ear spaces, often with adhesions and polypi. Acute flare-ups may occur with each head cold or from getting dirty water in the ear. Many of such cases resist all conservative treatment. A number of cases of Pseudomonas aeruginosa (Bacillus pyocyaneus) infection belong in this group. Conservative treatment is to be persisted in as long as hearing is serviceable; if the opposite ear is very deaf, as long as any hearing is present. We are not now talking about cases of threatened complication. But, when in these cases the radical procedure is done, it should be rather called a radical mastoidotympanectomy, for here the middle-ear work is of prime importance. Attention must be given to the Eustachian-tube region, the peritubal cells, hypotympanic cells, and subcochlear cells, if present. The floor of the external auditory canal should be lowered flush with the floor of the middle-ear cavity. Any drum remnants and muscle fragments must be removed. The mastoid bowl must be kept as small as possible if it is sclerosed.

Split-thickness skin grafts to the radical cavity have greatly shortened the time of healing. A dry ear occurs sooner than if the cavity is left to heal by granulation tissue.

When the middle ear is the seat of the disease, it is obvious that an incomplete radical procedure, let alone a simple mastoidectomy, will not reach the locus of the disease. As with the other procedures, every effort must be made to clear up infection reaching the middle ear by way of the Eustachian tube. The work of Guild [2] has shown the direct communication of peritubal cells with the Eustachian tube, and for this reason we feel that curettage of the Eustachian tubal opening should be done whenever pathology is encountered here. The accurate placing of a split-thickness skin graft over the tym-

panic opening of the Eustachian tube will greatly help to accomplish a dry ear.

In cases of complications supervening upon a chronic otitis, the aim is not to produce a dry ear primarily but to treat the complication. Frequently dural plate bone or lateral sinus covering will have to be removed. Some form of labyrinthectomy may have to be done in addition to the complete radical mastoidectomy.

The detailed treatment of such complications as facial paralysis, labyrinthitis, extradural abscess, and brain abscess is not within the scope of this paper, but will be made the subject of another paper.

SUMMARY

The various surgical procedures for the treatment of acute and chronic temporal bone infection are reviewed, with special emphasis on their indications, purposes, and aims.

The use, and sometimes the misuse, of chemotherapeutic and antibiotic agents has changed the symptomatology of middle-ear and mastoid infections.

Extensive pathology may go untreated until deafness results if one relies on the old criteria for surgical intervention.

An indicated surgical procedure should be timely if best results are to be expected.

The indications for myringotomy are given. If one relies exclusively upon the antibiotics for all cases, some will result in deafness.

The simple mastoidectomy should be as radical as possible, and the radical mastoidectomy, as simple as possible.

The modified radical mastoidectomy is indicated to preserve hearing by the prevention of further chronicity already started.

Aural polypectomy with cauterization of granulation tissue is sometimes preferable to radical mastoidectomy.

Ossiculectomy has been replaced by the modified radical procedure.

The complete radical operation varies in its requirements as the type and extent of pathology present and should be adapted to suit the needs of the individual case.

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The Surgical Treatment of Vertigo

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The surgical procedure on the labyrinth for the relief of vertigo is at present limited to cases with unilateral labyrinthine involvement and, perhaps with exceptions, to those without preservable hearing in the affected ear.

The indications for surgical treatment could, however, be considerably broadened if an excellent method could be found to destroy the vestibular function or at least enough of it to eliminate the vertigo and at the same time preserve or even improve the hearing. The indications would then also include the by no means rare bilateral involvement of the labyrinth and the frequent cases with usable hearing in the affected ear.

The possibility that this aim could be achieved is suggested by the fact that after coagulation of the labyrinth (Day's operation) the hearing is preserved and in some cases even improved beyond the preoperative level. It is, however, at present impossible to know beforehand in which cases this result can be expected.

Day and Lindsay [1] reported one case in which a coagulation of the labyrinth had been performed five years before death and a postmortem histological examination was done.

Day [2] later reviewed his experiences in 54 cases in which he had carried out a destructive operation on the labyrinth.

He had used his coagulation technique in the earlier cases but in later cases employed a dental broach to destroy the vestibular structures, followed by coagulation with a fine needle electrode.

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Altman [3] reported 15 cases in which coagulation of the membranous horizontal canal was performed. Vertigo was relieved in all; hearing was completely lost in 11 and decreased in 4, and tinnitus was unchanged in 5 but disappeared or was improved in 10.

In order to obtain additional information, operations were carried out in two cases.

REPORT OF CASES

CASE 1.—A 39-year-old patient complained of intermittent tinnitus in the left ear for the past five months, diminished hearing in the same ear, and attacks of vertigo for the past four months. A diagnosis of Ménière's disease was made. The attacks of vertigo persisted in spite of medical treatment.

Audiometer tests showed normal hearing in the right ear and reduced hearing of varying degree in the left ear (Fig. 1).

Turning and caloric tests revealed a moderate hypofunction of the left labyrinth, with normal function on the right side.

The electric goniometer test (Honjo-Furukawa) showed a moderate hypofunction of the labyrinth (Fig. 2).

On May 30, 1955, coagulation of the left labyrinth was performed. After exposing and making a fenestra into both the horizontal and the superior semicircular canal ampullas, the needle was introduced into the vestibule and its contents were coagulated.

During operation nystagmus was present, which at first was horizontal-rotatory to the side operated on and thereafter to the opposite side.

One week after operation, there was complete loss of hearing, which remained permanent. There was a slight unsteadiness after the operation for a few weeks, and it has persisted to the present. One month after operation, tinnitus was unchanged.

CASE 2.—A 47-year-old patient had had incapacitating attacks of vertigo and severe headache for one and a half years after aural trauma, with decrease in hearing and tinnitus in the left ear. The attacks of vertigo persisted in spite of medical treatment.

The audiogram showed a uniform hearing loss in the left ear, averaging 70-80 db., with near-normal hearing in the right ear (Fig. 3).

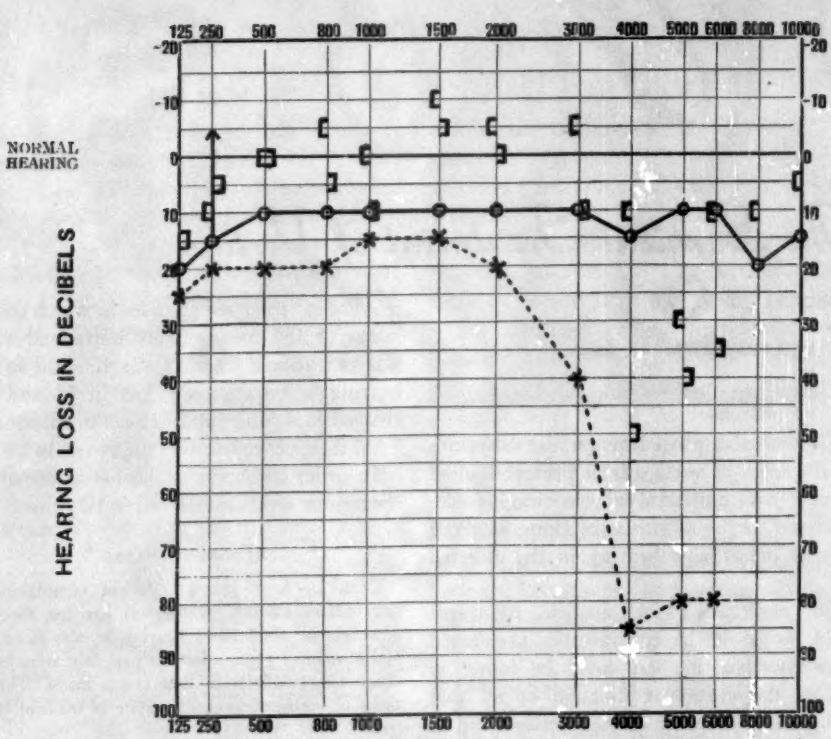

Fig. 1 (Case 1).—Audiogram. O——O, air conduction, right ear; X----X, air conduction, left ear;], bone conduction, left ear; [, bone conduction, right ear.

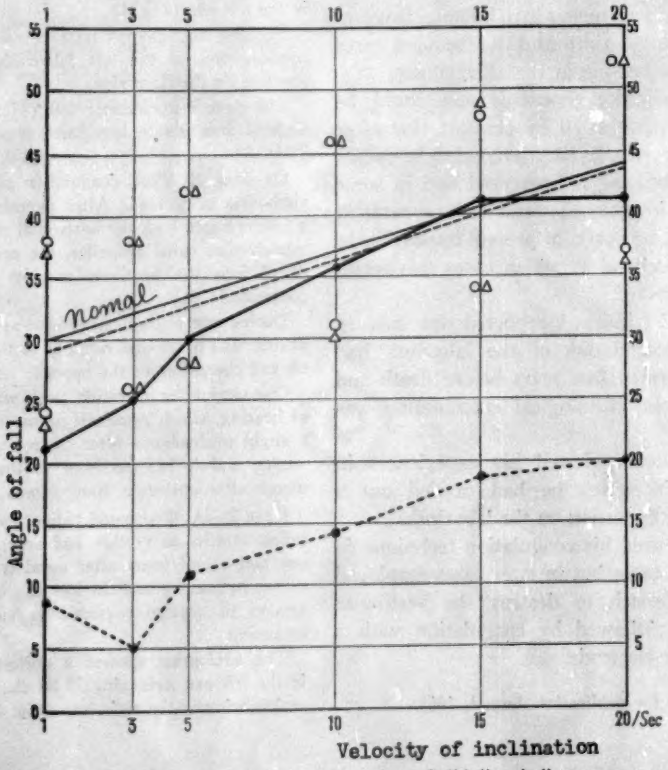

Fig. 2 (Case 1).—Goniogram taken before operation. Solid line indicates open eyes, and dotted line, closed eyes.

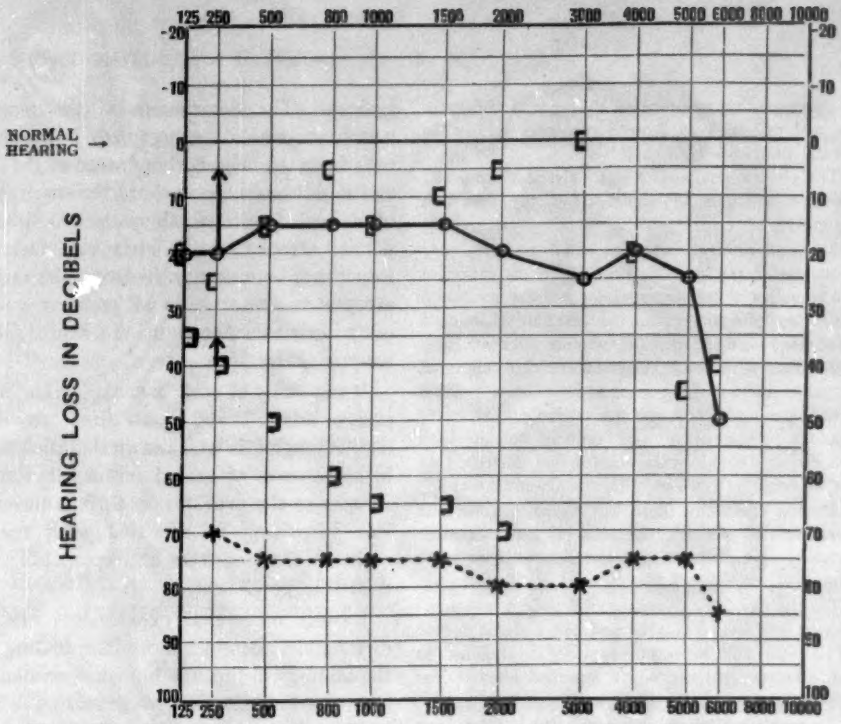

Fig. 3 (Case 2).—Audiogram. O——O, air conduction, right ear; X- - - -X, air conduction, left ear;], bone conduction, left ear; [, bone conduction, right ear.

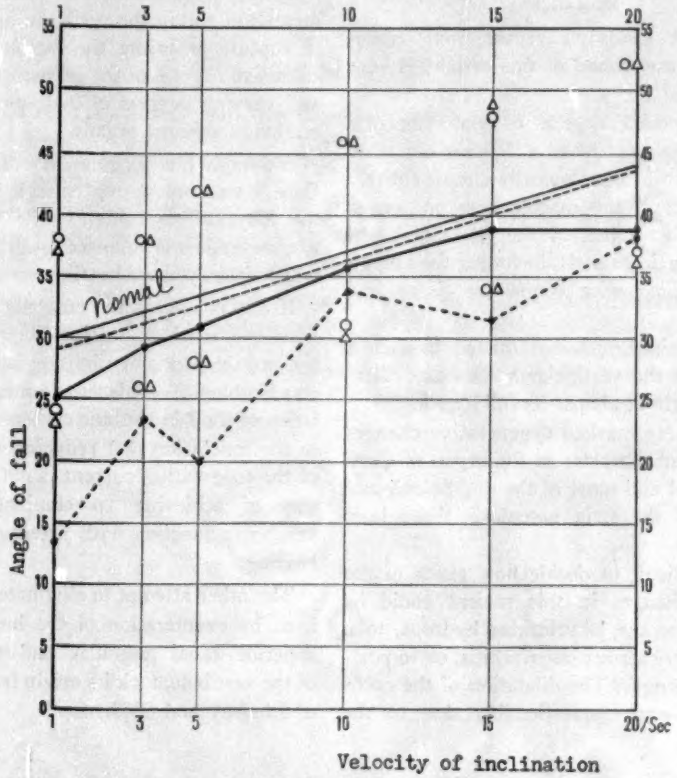

Fig. 4 (Case 2).—Goniogram taken before operation. Solid line indicates open eyes, and dotted line, closed eyes.

There was no spontaneous nystagmus, and turning and caloric tests showed responses within the normal limits for both ears.

The electric goniometer test (Honjo-Furukawa) revealed a slight hypofunction of the labyrinth (Fig. 4).

Since all medical treatment had been completely unsuccessful, on May 30, 1955, the exenteration of the ampullas of both the superior and the horizontal canals and the contents of the vestibule (Lindsay's operation) was performed by making a fenestra into the horizontal and the superior semicircular canal ampullas and enlarging these so as to form a single broad opening leading into the vestibule. The membranous structures in the vestibule and in both semicircular canals were ablated and destroyed as far as could be reached with dental excavators.

During operation, there was nystagmus, at first horizontal to the side operated on and thereafter rotatory-vertical to the opposite side, without vomiting.

On the day after operation, the patient was completely free from headache and had a slight degree of vertigo and horizontal-rotatory nystagmus to the side operated on, which subsided after a few days; the tinnitus remained unchanged.

Twenty days after operation, the hearing was completely lost.

COMMENT

Day and Lindsay's report (one case), previously mentioned in this article, is here reconsidered.

An advanced degree of endolymphatic hydrops was present in a 56-year-old man. The utricle was histologically almost entirely destroyed, and the macula was no longer present. The lumen of the horizontal and superior vertical canals, including the ampullas, was filled with connective tissue and new bone.

The cochlear duct was dilated to such a degree that the vestibular scala was obliterated throughout almost its full extent.

There were marked degenerative changes of a peculiar character in the organ of Corti in the basal and most of the middle coil and atrophy of the stria vascularis throughout the cochlea.

It is difficult to decide how much of the cochlear changes in this patient could be attributed to the labyrinthine hydrops, to a postoperative serous labyrinthitis, or to postmortem changes. The dilatation of the cochlear duct was unquestionably due to the hydrops. The detachment of the tectorial membrane and its overgrowth by epithelial cells from the neighborhood, seen in the basal and middle coils, seem characteristic features of a healed serous labyrinthitis. Although partial atrophy of the stria vascularis has sometimes been seen in hydrops, the marked atrophy of the stria in all cochlear coils is more likely to be the sequel of a healed diffuse serous labyrinthitis.

It can safely be said that coagulation in this patient was followed by a diffuse labyrinthitis. Although it is well known that the bearing in some cases of healed serous labyrinthitis returns to the previous level, it is nevertheless surprising to see that such marked cochlear changes were still compatible with relatively good hearing.

Results of animal experiments are, unfortunately, not much more illuminating than the findings in this one human specimen that has come to postmortem examination.

Proctor experimented on rabbits before penicillin was available and found that instrumentation within the vestibule in an attempt to rupture or injure the utricle resulted in extensive fibrosis of the perilymphatic spaces and varying degress of hydrops and degeneration of sensory organs.

Although the coagulation current in our Case 1 was not strong enough to coagulate the labyrinthine contents, the intervention was nevertheless followed by a labyrinthitis which destroyed the hearing completely.

If one compares the outcome in our case with the marked hearing improvement in the case of Day and Lindsay, in which there was histological evidence of extensive coagulation of the labyrinthine contents, one comes to the conclusion that reducing the intensity of the coagulating current is not an effective way of achieving an elimination of the vestibular function with preservation of the hearing.

The other attempt to eliminate the vertigo, i. e., by exenteration of the horizontal and superior canal ampullas and the contents of the vestibule, took its origin from the claim of Lindsay and Siedentop.[4]

Lindsay and Siedentop operated by making a fenestra into both the horizontal and the superior semicircular canal ampullas and enlarging these so as to form a single broad opening leading into the vestibule. The membranous structures in the vestibule and in both semicircular canals were ablated and destroyed as far as could be reached with suitable hooks, forceps, and dental excavators. The results have up to the present been uniformly good. Hearing has been totally destroyed, the attacks of vertigo have been successfully prevented, and the tinnitus was relieved in all cases. The follow-up periods have been six months to two years.

SUMMARY

Two cases in which labyrinthine surgery was used in treatment of vertigo in our clinic are reported.

Our experience shows that coagulation of the labyrinth fails to eliminate the vertigo and preserve the hearing.

Therefore, we feel that more radical surgical destruction of the labyrinth, i. e., exenteration, should be used.

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News and Comment

ANNOUNCEMENTS

Course in Laryngology and Bronchoesophagology.—The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period March 5 through March 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk St., Chicago 12.

Meeting of Eye, Ear, Nose, and Throat Societies.—Thursday, March 15, 1956, the Omaha Eye, Ear, Nose, and Throat Society and the Nebraska Academy of Ophthalmology and Otolaryngology are holding a joint all-day session. The principal speaker for ophthalmology will be Dr. Harold G. Scheie, of Philadelphia, and the principal speaker for otolaryngology will be Dr. George Shambaugh Jr., of Chicago. Members of the neighboring Eye, Ear, Nose, and Throat Societies have been invited, and members of the Kansas City Eye, Ear, Nose, and Throat Society have made plans to attend as a group.

A New Formula for Using the Audiogram to Predict Speech Hearing Loss

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INTRODUCTION

During the past three decades three trends in tests of hearing have proceeded simultaneously: first, a growing dissatisfaction with the spoken-voice and whispered-voice tests given since time immemorial; second, a growing intrusion of the commercial audiometer into the testing situation. Some have condemned the free voice tests as only the roughest of screens, neglecting their value in the hands of a really experienced tester, and others have looked to the audiometer as science's answer to all hearing testing problems, neglecting the difficulties of calibration, administration, and interpretation which may make results quite misleading.

A third trend has also proceeded somewhat more recently, in the development of the so-called speech audiometer. Although some few research-minded otologists have been using the phonograph for speech testing for almost 50 years, electronic speech-amplification systems calibrated for speech audiometry are still much rarer than the pure-tone audiometer, now in use by the thousands.

At the present time in this country standardizing bodies have specified the equipment necessary for either pure-tone [1] or speech [2] audiometry, and research has in general shown that acuity measures using either type of sound can be made with comparable reliability. The implication is that one can now have available on any patient either or both measures, without prejudice to either on the grounds of reliability. The question would then seem to become which to choose.

It is our thesis that the matter need not always, if ever, come to a choice. It is certain that the pure-tone audiometer, especially including the high frequencies, a well-calibrated bone vibrator, and provision for recruitment testing, gives vital information not obtainable with any speech testing. On the other hand, the importance of speech being preeminent in our civilization, nothing will really satisfy the practical question of how well a patient can hear speech except a reliable and bona fide speech test.

The only way to assess completely the acuity of an ear, then, is to give both an extended-frequency-range audiogram and a speech test in addition.

A. ARE SPEECH AND PURE-TONE TESTS EQUIVALENT?

There remain several reasons why it becomes important to examine quite closely the more exact relation between acuity for pure tones and for speech. In the first place, for theoretical reasons we wish to know whether the two types of test are really measuring different primary abilities, much as the broad area of intelligence has been broken down by psychologists into a number of primary men-

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tal abilities. This is a distinct possibility, as seen from the rather low intercorrelations of many psychoacoustic tests. If, on the other hand, we can establish that at threshold the two types of test, pure tones and speech, really measure the same ability, the correspondence only being subject to the many variables added by the pathological or otherwise unusual case, then there will be many situations where time and energy can be saved by eliminating either speech or pure-tone testing. Such situations might be in screening largely normal populations, such as school children or military inductees, or perhaps patients with conductive deafness and flat audiograms for whom (except perhaps for the severely deaf or the newly fenestrated otosclerotic) the speech acuity can with considerable confidence be predicted.

Again for theoretical reasons, it is important to examine the speech-tone hearing problem in order to assess whether we have a correct notion of how speech is perceived. Other things than pure-tone acuity are certainly involved. We do not know how much frequency contributes to speech intelligibility, how much is contributed by frequency discrimination, how much intensity discrimination contributes. We cannot well predict from the pure-tone audiogram whether a distortion will appear in the speech intelligibility function. We have little supporting research for the absolute values of Fowler's correction factor for recruitment. From a statistical standpoint, we do not know how much of the variance of speech tests is caused by variance in the measures of acuity for pure tones. Light will be thrown on these relationships only by examining speech-tone hearing discrepancies on many patients with many varieties of disorder.

B. The Medicolegal Problem

A legal complication arises also in assessing percentage disability. Should one use a speech test or a pure-tone audiogram? The American Medical Association uses the audiogram, converting by a roundabout way to percentage loss for speech. The Veterans Administration uses the speech audiometer directly. Different states use different systems. Until agencies can generally agree upon an actual speech test it will be necessary, for some time to come, to provide a conversion from pure-tone data to a predicted speech score.[8]

C. The Clinical Importance of Real Discrepancies

One last major reason exists, from the clinic, to reevaluate the speech-tone problem. There are cases of real discrepancy between hearing for the two types of material, and these cases must be looked at with special care. Some merely need referral to the clinical psychologist, the neurologist, the speech correctionist, etc. But in others, the discrepancy may be useful in the diagnosis. An accurate index of the amount of this discrepancy may then become important. Unfortunately, at present the estimation of such discrepancy may depend entirely on what system is used for compressing the audiogram into a predicted speech score. The several systems described below are based upon different fundamental principles—all differences are not simply in the weights assigned to the several audiometer frequencies—and for some patterns of pure-tone loss will predict quite different speech scores. These different speech scores may differ from each other by as much as 50 db. or more, and the actual speech may lie somewhere between or may be close to one prediction and far from the others. Examples of these confusions will be made explicit later in this paper. We wish here to point out only that solely because of a lack of agreement among predictive systems it is at present impossible, for some ears, to say whether a speech-tone discrepancy does or does not exist.

COMMENT ON PREVIOUS SYSTEM

Inasmuch as quite wide divergences appear with the use of the predictive systems in current use, and since no very recent broad critique of all these exists in the literature, it seems in place here to examine the theoretical bases on which each has been built up and to compare each with what is now

known about the psychophysics of speech hearing.

A. The Three-Average System

A good portion of the effort of Fletcher and his many colleagues at the Bell Telephone Laboratories has been devoted to just this question: the relation between acuity for pure tones and for speech. Using as a basis studies of speech audibility and intelligibility with various frequencies filtered out, and examination of speech spectra, Fletcher determined that the region 500 through 2000 was especially important in speech hearing. This has been confirmed many times over, and his suggestion in 1929 that the average audiogram through this range be taken as the best prediction of speech hearing is still very widely followed, and works as well or better in practice as any other system to date.[4]

B. Sabine's System

A very different system was proposed by Sabine,[5] based on the relation between speech reception and the ability of the ear to make psychoacoustic discriminations (for pitch and loudness) over the frequency regions and intensities found in speech. Sabine started by accepting Bunch's estimate of the decibel loss at each frequency for total loss for speech. The auditory area between these losses and zero on the audiometer was then divided up into blocks one octave wide between 128 and 4096 cps, and 10 db. high, and credit assigned to each block according to the percentage within that block of the total number of distinguishable intensity differences over the whole audiometric area. The number of distinguishable frequency differences was said to be very similar. Thus the percentage of ability to understand speech, according to Sabine, depends directly upon the number of discriminable frequency and intensity steps available to the patient. Two additional points were made: that speech emphasizes certain frequency regions and that the percentage of speech sounds heard correctly is not a linear function of intensity. Accordingly, the weights in each block were in a second version rather arbitrarily changed to accord with the Bell Laboratory studies on frequency filtering, and also to accord with the Bell studies on speech intelligibility as a function of intensity. Thus Sabine's final weights were based upon a supposed relation between speech reception and pitch-plus-loudness discriminations, modified to allow for the special importance of the 500 to 2000 band, and to allow for the sigmoid shape of the articulation curve.

It is, in our opinion, erroneous to weight the various intensities differently according to the shape of the articulation curve, as Sabine did. We reason that the significant point in a speech test is the 50% intelligible intensity (or whatever other intensity, as the 75% or 95% intelligible intensity, may be the convention of the moment). But the difference between this intensity for a normal and for many defective ears is not at all dependent upon the shape of the articulation curve. These defective ears yield articulation curves very similar to normals, only displaced at the 50% intelligible intensity by approximately the amount of pure-tone deafness on the audiogram. This is true for many perceptive-deaf as well as conductive-deaf patients, as our material shows in this paper. It is, of course, true that in the normal ear to increase speech intensity from 50 to 60 db. re 0.0002 microbar is to improve intelligibility only 2%-3% because of a ceiling effect; but of two patients with audiometric losses of 50 and 60 db., there will be differences in intelligibility of 0%-40%, depending entirely upon the intensity level of the speech sample. It is therefore unjustified to say that the shape of the articulation curve should determine the vertical weights on the audiogram.

For a determination, then, of the decibel loss for speech, each 10 db. increment of intensity should carry the same weight, since for all comparisons between normal and defective ears we are working on the same point on the articulation curve irrespective of its shape (with the exception noted, of those ears which show definite distortion of this curve).

Sabine's system had importance, since it was adopted almost in toto by the American Medical Association in 1942.[6] One could wish, however, that the assumptions on which the Sabine system rests were better documented. The original weights for each block of one octave × 10 db. depend upon speech reception actually being a function of psychoacoustic discriminations of pitch and loudness. But although at first glance this looks to be a truism, it is not certain how close the relation really is. It may be, for example, that speech intelligibility depends far more on its temporal pattern than on the very precise discriminations of pitch and loudness of which the human ear is capable. We know that those ears with very poor pitch discrimination can often handle speech very well. Furthermore, the frequency component of speech can be entirely removed, as in square-wave speech, or distorted badly by introducing a large amount of frequency modulation or "wow" in the playback system, yet intelligibility is not completely destroyed.

C. The Loudness-Loss System

We have investigated the possibility of using the one octave × 10 db. block system, just as Sabine did, but, instead of using the number of psychoacoustic discriminations, we calculated the percentage loudness, in millisones, contributed by that block in comparison with the whole speech area. This possibility, however, leads to a cul de sac on both theoretical and practical grounds. Using Fletcher and Munson's graphs [7] the millisone contribution of each block was easily calculated; but a fundamental difficulty arises, in that the number of millisones a defective ear cannot experience, in comparison with a normal ear, bears no simple relation to the decibel difference between the acuities of the two ears. A transformation could be constructed using a loudness scale, but no final agreement exists at present as to the precise shape of such a scale, and in any case it is known that individual differences in loudness scaling would, for an individual patient, render prediction of his speech loss quite impossible.

In spite of these fundamental difficulties, but especially because they are no more than those Sabine wrestled with, we did take the step of constructing a table of blocks within the auditory area for speech, each block containing its percentage contribution to the total number of millisones. But, as expected, the contribution of the blocks up to 30 db. over threshold was so negligible (only 1.8%) that the idea was clearly indefensible. In order to correspond either to decibel loss for speech or to percentage loss, the weights would have to be altered both horizontally and vertically beyond recognition.

We have now exhausted the principles of psychoacoustic discriminations and of loudness as bases for speech *vs.* audiogram comparisons. Other principles could be examined, such as the ability of the ear to perform temporal integration or to make temporal discriminations, etc., over the speech area. But these notions demand more capital than our research bank today possesses before they could pay off in the clinic. Our best bet of the moment seems to be to use acuity for pure tones as a direct predictor of acuity for speech, rather than to go through some intermediary concept such as loudness. There have been several attempts to use the audiogram directly to predict speech hearing loss, in addition to Fletcher's original Three-Average system. All of these assign different weightings to the various frequency regions.

D. Fowler's 1941 and 1942 Systems Based upon Empirical Observations

No one has worked harder than Fowler to establish a method for predicting hearing loss for speech using only pure-tone data. In 1941 [8] he gave full details on a system empirically worked out on his many hundreds of patients, all patients carefully assessed from just the point of view of speech-tone correspondences. As applied to a single ear only, it assigns weights of 15, 25, 30, 25, and 15 to the audiometric losses at 500, 1000, 2000, 3000, and 4000 cps, respectively; one multiplies the pure-tone losses by these weights, takes a total, and divides by 100.

The result is the weighted decibel loss for speech for that ear.

Adjustments are made for ac-bc discrepancies indicating the presence of recruitment: up to 4 db. credit if the deafness is 35 db. or less, subtractions of up to 10 db. if the deafness is over 40 db.

Subsequently [9] the weights were simplified to 15, 30, 40, and 15 at 500, 1000, 2000, and 4000 cps, the limit of weighted decibel loss was dropped from 110 to 100, and the recruitment factor adjustment limited to a 5 db. maximum. This latter system Fowler still advocates [10] as corresponding best of any system to his patients' real capacities for handling all kinds of speech.

The weighted decibel loss for speech for any ear was then converted into percentage loss for speech by the following figures:

Weighted Loss, Db.	Loss for Speech, %	
	1941	1942
10	0	2
20	5	9
30	14	20
40	27	35
50	44	54
60	62	71
70	77	84
80	88	93
90	95	98
100	98	100
110	100	..

This tabulation represents an experienced otologist's best guess as to the relative importance to daily living of ability to hear very weak, medium, and very loud voices. We concern ourselves here only with the fact that the phrase "percentage loss" means something very different in the Sabine and in the Fowler systems.

Fowler's system is not easily open to a theoretical discussion, since it is not based upon a general theory of how the ear hears speech. Weights were tried and discarded until the latest proposal was evolved, quite on a trial-and-error basis. This criticism of the system has no practical validity, however, to the patient nor, indeed, to the clinician. If Dr. Fowler has evolved a system which correctly correlates speech and pure-tone hearing, then theories of speech hearing must be constructed or changed to take this correlation into account. The only approach to check the usefulness of Fowler's system is to try it out on large populations and to compare it with systems based on different principles or weights.

The importance of Fowler's most recent weights is that they have been adopted by the American Medical Association.[11] Let us now look at the A. M. A. 1947 system and see what became of Sabine's concept of percentage loss as depending upon the number of psychoacoustic discriminations remaining to the defective ear. A comparison of the horizontally added weights shows that the original figures derived solely from the numbers of psychoacoustic determinations available have suffered a sort of sea change. First altered by Sabine to conform to the shape of the articulation curve, they were modified further in 1942 by the A. M. A. Committee in a sort of compromise between Sabine's and Fowler's proposed weights, and, again, by the A. M. A. Committee in 1947 to such an extent that they differ by never more than 2 db. from Fowler's final 1942 weights, as given in the tabulation above. Thus the most recent A. M. A. system no longer represents percentage loss in terms of number of psychoacoustic discriminations but has been converted entirely to represent Fowler's figures for percentage loss in terms of general capacity to hear all types of speech.

A similar conversion has likewise changed the weights for the several frequency regions. The weights for the frequencies now used by the A. M. A., 500 through 4000 cps, are almost exactly 15, 30, 40, and 15 at all intensity levels, again in exact accordance with Fowler; but the weights in the original table, unmodified by Sabine, differed at various intensities. For example, at the 30 db. level the weights beginning at 250 cps were 13, 13, 13, 30, and 30, and at the 70 db. level the weights were 13, 16, 19, 26, and 26. These original weights were modified by Sabine on the basis of frequency-filtering data, and twice more by the A. M. A. to conform finally with Fowler's ideas on the

relative importance of frequency regions in speech communication.

E. Fletcher's 1950 System

Although his original Three-Average system had good success, Fletcher early felt that some consideratoin should be given to a differential weighting of the frequencies involved. Having worked out in an epochal experiment a way to calculate the loudness, in sones, for sounds of any frequency composition, Fletcher had only to make the appropriate measurements for actual speech: (1) the intensity level at the threshold of audibility (not intelligibility) for a normal ear listening to speech filtered through very small frequency bands and (2) the spectrum level of conversational speech in those same small bands. With these two sets of data, the loudness of speech at any level could be ingeniously calculated for the normal ear. Now Fletcher made the reasonable assumption that the loudness loss in speech, caused by a defective audiogram, was equal to the loudness experienced by a normal ear if the speech were raised as many decibels as the audiogram is defective. (Because of the use of small frequency regions in measurement, it is possible to allow for a differential raising of intensities at various regions, thus simulating distorted audiograms. This is important in the system, since some frequency regions contribute more to the loudness of speech than other regions.)

By means of the assumption referred to above, Fletcher is able to make the jump from loudness data on normal ears to decibel loss for speech in the abnormal ear.[12]

Two or three points must be emphasized in considering the application of Fletcher's weights to the hard of hearing patient: 1. The whole system is concerned with the loudness of speech. This basic principle seems in general well founded, but, of course, it has been emphasized many times, especially for perceptively deaf ears, that the loudness of speech is not always a good index of its intelligibility. 2. The weights assigned to the different frequencies come partly from measurements of average total speech power; but the power in speech is largely a function of the vowels, whereas th c ns n nts c rry th nt ll g b l t. 3. To find the estimated hearing loss for speech, in decibels, the following formula is used:

$$HL_{speech} = -10 \log \left(8.30103 - 10^{-\frac{HL_{500}}{10}}\right) + \left(9.20412 - 10^{-\frac{HL_{800}}{10}}\right) + \left(9.60206 - 10^{-\frac{HL_{1000}}{10}}\right) + \left(9.60206 - 10^{-\frac{HL_{2000}}{10}}\right) + \left(8.30103 - 10^{-\frac{HL_{4000}}{10}}\right).$$

One adds the antilogs of the five parenthetical terms, finds the log of this sum, and multiplies by —10. The calculations for each audiogram may be avoided by previously making up a handy table.

But an inspection of this formula will reveal that an audiogram will never yield a predicted speech score much worse than the average loss at the two best of the five frequencies. Yet, thus heavily weighting the best-heard frequencies is often wide of the mark, especially for one of the commonest audiometric patterns, a sloping high-tone loss. Consider an actual ear, normal at 250 but sloping off at the higher octaves in amounts of 15, 40, 45, 55, and 60 (Ear 18, Table 1). Most systems will predict speech losses of 30 db. or more, and his actual speech loss is 36 db. But the Fletcher 1950 system, leaning heavily upon the two best frequencies, predicts only a 16 db. loss. When speech sounds sampling the high-frequency components are used for testing, the Fletcher 1950 system will systematically underestimate the hearing loss in cases of progressive high-tone hearing loss.

F. Fletcher's Two-Best System

A short method was proposed at the same time by Fletcher[12] to take the average of the hearing losses at the two best of the three frequencies 500, 1000, and 2000 cps. This method suffers from some of the objections already cited to the Fletcher 1950 system.

G. The Two-Worst System

It might even be proposed that the average of the two worst of these frequencies be taken, on the grounds that the distortion pro-

duced by the one worst frequency (disregarded, of course, in the Two-Best system) may well need to enter the picture.

H. Fletcher's 1953 System

Another system has been proposed by Fletcher [18] with still different frequency weights, based upon loudness concepts. For the octave intervals from 125 through 8000 cps the weights in order are 0.003, 0.037, 0.13, 0.25, 0.30, 0.23, and 0.05. The sum of the hearing losses multiplied by the respective weights gives a weighted decibel loss for speech hearing.

It must be mentioned parenthetically here that Fletcher proposed that these weights contribute to a "percentage loss" for speech rather than to a weighted decibel loss. The hearing loss at each frequency was to be divided by an amount, between 75 and 105, depending upon frequency, for values of which the ear could be considered totally deaf. In this system, therefore, one encounters still a third meaning for "per cent loss of hearing," based on the assumption that per cent loss is some linear function of decibel loss. For the purposes of the present paper, we disregard this use of per cent loss, as well as the method in this system for combining data from the two ears, and we simply use the sum of the weights given above when multiplied by the audiometric losses.

I. Multiple Regression Equation System

Still another predictive system is possible, based on no hypothesis as to how the ear perceives speech, but based entirely on statistical principles. The problem of prediction which we meet here is one found commonly in work with the vastly complicated human being, where no two variables are ever related in a simple manner. Special techniques have been developed for the situation where a criterion variable (in this case the actual speech score) is a function of several predictor variables (in this case the audiometric losses at the several frequencies). We ask: What weight should be assigned to each of the predictor variables in order to obtain the best estimate of the criterion variable?

A statistical technique to answer just this question has been well worked out; it is the multiple regression equation, derived from partial regression coefficients. The reader who wishes an understanding of these concepts is referred to any advanced statistics text. It is enough to say here that a partial regression coefficient comes from the relation between, in our case, hearing loss for speech versus hearing loss at one audiometer frequency, with the influence of all other frequencies canceled, that is, rendered inoperative by statistical means. The sense of this is apparent when one takes a specific example, such as the high correlation between hearing loss for speech and for loss at 250 cps. The fact is that this high correlation arises not because of a close dependence of speech on 250 cps but simply because 250 cps correlates highly with 1000 cps, upon which speech is dependent. In this case, the unduly high speech 250 cps correlation must by special controls be kept from influencing one's final interpretation.

The partial regression coefficients for each frequency are used as weights in the final multiple regression prediction equation. Hearing losses at each relevant frequency are multiplied by their respective weights, added, and the sum is the prediction of the speech hearing losses for those persons comprising the whole group of patients from whom the original data were derived. (See Appendix.)

The only real question is whether the equation will work for other speech materials and other groups of patients. Here one can strive for generality only by including a representative variety of types of deafness, ages, audiometric patterns, speech materials, etc., in the original study. If this is done, the equation will prove continually useful.

It is entirely possible with this technique to write more than one equation, perhaps one for conductive deafnesses with flat audiograms, another for sloping high-tone perceptive deafnesses. Certainly different equations would best fit spondee and PB speech scores.

But for any one situation, the worker could be assured that his prediction equation would produce the least possible total error.

PROCEDURES OF THE PRESENT STUDY

In view of all the foregoing, it seemed worth while to us to attempt a direct comparison of the several possible systems for predicting speech hearing loss using only pure-tone data.

To this end, a series was collected of 197 partially deafened ears (the patients aged from 12 through 58) of a wide variety of diagnoses, the widest variety of audiometric patterns, durations of disability, etc.

On all of these ears both air- and bone-conduction audiograms were collected with scrupulous attention to the details of calibration, masking, etc., as well as complete articulation curves with the C.I.D. W-22 PB records. All ears were examined with the otoscope, and a complete audiological history was obtained. Loudness recruitment and pitch discrimination were tested on several dozen ears. For many of the ears, and indeed all for which the issue was in doubt, an otological examination and diagnosis were obtained. It was almost always possible, to our no small comfort, on the basis of the many indices available, to categorize any loss as perceptive, or conductive, or mixed—largely perceptive, or mixed—largely conductive, or mixed—about equally perceptive and conductive; for a few ears it seemed possible to assign a nonperipheral basis to the perceptive involvement.

It should be pointed out that our population was not composed solely of relatively young servicemen. About half our patients were not servicemen. There was, however, somewhat more noise trauma and somewhat less presbyacusis and severe childhood hypacusis than might be found in the usual hearing clinic. For the most part, however, our group well represents the types of patient one sees in the usual ENT practice in a small city.

RESULTS

The bulk of our data can be found in Tables 1, 2, and 3. The column headings

TABLE 1.—*Data from Sixty-Four Ears with a Discrepancy of Twenty Decibels or More Between 500 and 2000 Cps, by Air Conduction*

Ear	250	500	1000	2000	3000	4000	6000	8000
1	5	5	40	25	30	40	80	60
	10	10	40	25	30	40	..	..
	28	28	27	12	31	15	30	..
2	0	0	0	50	45	60	50	50
	10	10	20	55	60	40	..	..
	20	17	31	2	32	0	24.4	..
3	0	0	0	50	50	60	40	35
	10	10	15	55	..	45	..	..
	20	17	31	2	32	0	23.7	..
4	5	5	10	60	60	55	65	60
	35	25	35	10	40	7	30.3	..
5	25	25	30	45	45	60	55	35
	15	20	25	30	40	60	..	..
	34	33	38	30	41	27	37.3	..
6	0	5	15	40	55	70	60	40
	0	15	25	40	55	60	..	..
	26	20	31	11	36	10	30	..
7	0	10	35	45	55	70	55	35
	5	20	30	55	60	60	..	..
	31	30	41	15	42	22	37.7	..
8	0	—5	0	20	75	90	..	..
	10	10	0	75	..	..	..	..
	8	5	21	0	32	—2	26	..
9	60	55	55	35	40	45	55	45
	25	25	20	30	35	35	..	..
	56	48	43	30	46	45	48	..
10	20	25	80	70	70	85	80	75
	25	35	..	..	..	..	..	..
	63	62	72	32	68	47	63.6	..
11	15	20	70	65	55	70	65	55
	20	30	..	..	..	..	..	..
	58	52	64	27	59	42	54.7	..
12	0	10	25	40	45	40	50	40
	10	20	20	50	35	40	..	..
	25	25	30	14	31	17	27.6	..
13	10	20	50	50	40	40	45	30
	15	25	35	40	40	45	..	..
	40	40	44	25	41	35	39.2	..
14	15	25	40	50	40	35	45	30
	15	30	30	35	40	40	..	..
	39	38	40	29	38	32	36.9	..
15	10	30	45	55	60	65	60	40
	10	25	40	55	50	..	..	..
	48	48	50	27	49	37	47.1	..
16	15	20	40	40	40	45	45	45
	25	25	35	50	40	35	..	..
	29	33	39	26	38	30	35.3	..
17	—5	0	20	30	35	45	60	65
	15	15	20	35	35	..	..	..
	21	17	24	11	28	10	23.7	..
18	0	15	40	45	50	55	60	60
	15	20	35	40	45	..	..	..
	36	33	40	16	41	27	37.9	..
19	—5	—5	0	20	10	15	10	5
	..	..	5	15	10	10	..	..
	12	5	8	1	10	—2	6.2	..

TABLE 1.—*Data from Sixty-Four Ears with a Discrepancy of Twenty Decibels or More Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
20	—5	—5	0	25	10	5	15	0
	..	..	5	30	15	0	..	..
	13	7	11	0	9	—2	5.7	..
21	45	50	45	25	25	30	30	20
	10	10	15	15	15	20	..	..
	36	40	31	29	35	35	37.4	..
22	5	5	70	60	60	55	55	15
	5	5	40	35	35	25	..	..
	31	45	53	13	49	32	46.4	..
23	—5	5	20	30	40	55	50	40
	5	10	20	30	..	50	..	..
	18	18	26	19	30	12	26	..
24	—5	5	20	35	55	60	60	50
	0	15	20	40	..	55	..	..
	23	20	28	19	33	12	28.7	..
25	—5	—5	0	25	50	50	50	50
	..	0	0	15	55	60	..	..
	22	7	17	1	22	—2	16.2	..
26	—5	—5	5	50	60	60	50	50
	..	0	5	40	60	..	..	..
	23	17	31	2	34	0	24.6	..
27	5	5	40	45	50	60	75	65
	10	10	30	50	50	50	..	..
	45	30	41	13	42	22	37.3	..
28	15	10	20	50	55	60	75	60
	15	15	20	55	55	45	..	..
	52	27	36	17	39	15	34.1	..
29	5	0	15	25	15	30	30	25
	..	..	15	15	10	25	..	..
	19	13	15	8	20	7	16.5	..
30	5	5	25	60	65	80	60	60
	15	10	25	55	..	..	..	..
	48	30	46	12	48	15	38.9	..
31	5	5	10	30	40	40	25	40
	10	10	10	45	..	50	..	..
	19	15	23	10	24	7	18.9	..
32	10	10	20	40	50	45	50	50
	15	15	25	50	55	45	..	..
	29	23	31	16	32	15	27.5	..
33	0	0	0	20	10	5	10	10
	..	..	..	10	..	..	..	..
	10	7	9	2	8	0	5.6	..
34	0	0	5	35	75	75	75	70
	5	5	5	40	..	..	..	..
	18	13	26	5	34	2	27.1	..
35	0	5	0	75	65	60	60	55
	10	10	0	50	..	..	..	..
	28	27	41	3	41	2	31.4	..
36	5	0	35	50	30	15	5	0
	20	20	35	45	40	20	..	..
	23	28	33	8	27	20	22.8	..
37	15	25	55	60	55	45	40	35
	20	25	40	50	60	55	..	..
	48	47	53	29	47	40	44.5	..
38	15	30	50	55	55	50	45	40
	15	20	45	45	60	50	..	..
	53	45	51	31	47	40	44.7	..

TABLE 1.—*Data from Sixty-Four Ears with a Discrepancy of Twenty Decibels or More Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
39	30	35	40	55	70	90	..	..
	30	30	35	45	..	..	..	..
	37	43	53	40	50	37	54.2	..
40	—5	0	15	25	10	25	15	5
	5	5	10	10	5	25	..	..
	23	18	18	6	17	7	14.6	..
41	5	5	5	45	70	75	70	65
	5	0	10	45	..	70	..	..
	15	18	33	7	37	5	30	..
42	0	5	0	35	50	50	50	30
	10	0	0	45	..	70	..	..
	9	13	24	3	25	2	20.6	..
43	5	10	35	30	20	25	60	30
	15	15	40	30	45	25	..	..
	23	25	26	16	26	20	26.8	..
44	10	10	40	30	40	50	70	55
	15	10	45	30	50	40	..	..
	24	27	33	17	35	20	33.4	..
45	5	0	30	50	45	50	65	46
	10	5	35	60	50	50	..	..
	32	27	39	8	37	15	31.8	..
46	5	10	45	45	45	50	70	50
	10	10	50	50	55	55	..	..
	41	33	45	17	40	27	37.7	..
47	80	80	100	100	100	100	..	80
	40	50	60	55	55	55	..	..
	105	93	101	87	96	90	93.1	..
48	0	0	10	20	10	5	20	0
	..	..	10	15	10	5	..	..
	14	10	10	6	10	5	9	..
49	—5	—5	0	20	25	50	50	50
	5	5	10	30	40	..	..	..
	11	5	15	0	21	—2	15.2	..
50	—5	—5	10	20	30	50	55	40
	5	5	5	35	35	40	..	..
	13	8	18	2	23	2	18.3	..
51	5	45	50	95	95	90	..	80
	10	35	40	40	50	50	..	..
	94	63	74	22	73	47	67.4	..
52	20	15	20	50	55	60	50	30
	10	5	20	40	60	60	..	..
	40	28	36	20	39	17	33.7	..
53	5	5	25	50	50	55	45	40
	0	0	25	40	50	40	..	..
	32	27	38	12	37	15	31.2	..
54	5	5	20	50	50	55	50	40
	0	0	15	45	50	40	..	..
	34	25	35	12	36	12	30.1	..
55	25	30	65	60	55	70	80	75
	40	45	55	..	..	..	..	..
	70	52	63	37	59	45	55.9	..
56	30	40	65	70	60	80	80	80
	45	50	60	..	..	..	..	..
	74	58	70	44	66	52	62.2	..
57	0	5	5	35	45	40	25	0
	10	5	5	30	50	45	..	..
	17	15	22	7	22	5	18.5	..

TABLE 1.—*Data from Sixty-Four Ears with a Discrepancy of Twenty Decibels or More Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
58	5	5	10	45	50	45	35	10
	10	5	0	40	30	40	..	..
	20	20	28	10	28	7	23.4	..
59	5	10	20	30	30	30	60	45
	10	20	20	25	20	30	..	..
	24	20	21	16	25	15	23.5	..
60	5	20	40	40	45	35	35	50
	10	20	25	35	40	40	..	..
	35	38	34	21	35	30	32.6	..
61	0	0	0	30	25	20	10	0
	..	..	0	30	25	20	..	..
	17	10	14	2	14	0	10.8	..
62	5	0	20	30	40	25	10	0
	..	..	15	30	35	20	..	..
	24	17	18	8	20	10	16.8	..
63	20	25	35	50	30	25	50	30
	0	5	5	35	15	10	..	..
	43	37	38	31	35	30	34	..
64	25	30	40	60	50	30	30	30
	5	5	5	40	25	15	..	..
	49	43	44	36	41	35	38.3	..

First row, air-conduction losses at frequencies indicated.

Second row, bone-conduction losses at frequencies indicated.

Third row, Column 1, speech hearing loss by PB list; Column 2, predicted speech loss by Three-Average system; Column 3, predicted speech loss by Fowler 1942 system; Column 4, predicted speech loss by Fletcher 1950 system; Column 5, predicted speech loss by Fletcher 1953 system; Column 6, predicted speech loss by Fletcher Two-Best system; Column 7, predicted speech loss by Multiple Regression prediction system.

TABLE 2.—*Data from Forty Ears with a Fifteen Decibel Discrepancy Between 500 and 2000 Cps, by Air Conduction*

Ear	250	500	1000	2000	3000	4000	6000	8000
65	10	10	20	5	5	20	20	5
	15	15	25	15	15	30	..	..
	1	12	8	8	13	7	13.6	..
66	—5	—5	10	10	10	10	10	—5
	3	5	7	3	4	2	6.1	..
67	20	20	35	20	10	15	15	0
	25	25	30	30	20	25	..	..
	7	25	21	22	21	20	22.4	..
68	0	0	10	15	10	0	15	10
	..	..	10	20	10	5	..	..
	10	8	7	6	7	5	6.7	..
69	0	5	20	5	0	0	15	5
	10	10	5	5	5	0	..	..
	10	10	7	6	9	5	8.7	..
70	0	0	0	15	50	50	45	40
	..	..	..	15	..	..	..	..
	11	5	12	2	17	0	15.1	..
71	5	5	15	20	20	30	25	15
	..	10	20	20	20	15	..	..
	12	13	14	11	18	10	16.4	..

TABLE 2.—*Data from Forty Ears with a Fifteen Decibel Discrepancy Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
72	5	10	15	25	25	15	25	15
	10	5	20	25	25	15	..	..
	15	17	15	14	17	12	18	..
73	0	5	15	20	15	40	25	25
	..	10	15	25	15	25	..	..
	15	13	15	11	21	10	18.2	..
74	0	5	10	20	5	15	10	0
	5	5	10	15	10	10	..	..
	16	12	12	9	13	7	11.4	..
75	10	5	30	30	85	95	80	70
	10	10	20	35	40	45	..	..
	17	15	28	12	30	12	23.3	..
76	0	5	15	20	35	55	55	60
	5	10	10	30	30	35	..	..
	17	13	20	11	27	10	22.9	..
77	0	0	10	15	15	10	5	0
	..	..	5	5	20	10	..	..
	17	8	10	6	9	5	7.5	..
78	0	0	15	10	30	40	40	0
	10	10	15	15	25	35	..	..
	17	8	11	6	16	5	16	..
79	0	5	20	15	0	0	—5	—5
	5	5	15	5	10	0	..	..
	18	13	11	10	10	10	9.4	..
80	5	5	10	20	35	35	45	25
	..	..	10	10	40	35	..	..
	18	12	17	10	19	7	18.4	..
81	—5	15	30	20	15	45	15	0
	5	15	30	20	30	50	..	..
	18	22	24	19	26	17	25.2	..
82	0	10	25	20	35	35	10	—5
	5	15	20	20	40	40	..	..
	19	18	19	14	22	15	22.3	..
83	10	15	25	30	35	45	25	5
	15	15	25	35	40	35	..	..
	20	23	26	21	29	20	26.4	..
84	5	5	15	20	20	10	20	25
	..	..	15	10	15	15	..	..
	22	13	12	11	14	10	12.5	..
85	0	0	0	15	20	20	15	0
	..	..	5	20	25	20	..	..
	22	5	7	2	9	0	7.6	..
86	10	10	15	25	15	55	80	80
	10	10	15	15	15	45	..	..
	22	17	23	15	30	12	26.9	..
87	20	25	35	15	20	45	50	40
	10	10	5	15	20	20	..	..
	24	25	24	18	30	20	30.5	..
88	5	10	15	25	50	50	35	25
	10	10	5	20	30	55	..	..
	24	17	23	14	26	12	22.9	..
89	10	10	30	25	50	65	80	80
	10	10	10	15	50	..	..	..
	25	18	26	16	34	15	30.2	..
90	5	20	25	35	40	50	20	—5
	10	15	30	40	45	50	..	..
	26	27	31	20	32	22	29.3	..

TABLE 2.—*Data from Forty Ears with a Fifteen Decibel Discrepancy Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
91	30	40	40	25	25	25	30	20
	40	30	40	30	30	30	..	..
	31	35	30	29	31	32	32.7	..
92	20	25	20	35	50	60	45	30
	10	15	20	25	40	50	..	..
	30	27	30	23	36	22	31.4	..
93	50	45	55	35	50	50	55	30
	20	20	15	20	25	25	..	..
	44	45	41	39	45	30	46.3	..
94	20	25	30	40	60	80	..	..
	..	..	..	..	..	..	..	..
	41	32	40	19	47	27	43.9	..
95	20	30	40	45	40	50	65	55
	10	15	25	40	45	50	..	..
	43	38	41	34	43	35	41.1	..
96	40	50	45	60	80	85	..	80
	35	35	40	50	..	..	..	..
	43	52	59	48	63	47	59.4	..
97	35	45	50	30	25	20	30	45
	5	5	10	20	20	5	..	..
	45	42	35	32	35	37	36.7	..
98	30	25	15	10	5	5	60	65
	20	15	10	5	5	..	..	..
	46	17	11	12	25	12	17.4	..
99	60	55	60	45	40	65	80	..
	25	20	30	20	25	40	..	..
	48	53	51	49	58	50	56.7	..
100	45	45	50	35	30	30	30	25
	5	5	10	20	20	20	..	..
	50	43	38	38	38	40	39.5	..
101	35	40	45	30	50	60	45	40
	10	5	10	10	40	40	..	..
	52	38	41	34	43	35	42.5	..
102	55	60	70	75	60	75	60	55
	15	15	25	25	25	25	..	..
	67	68	68	66	70	65	67.4	..
103	55	55	55	40	45	65	75	65
	10	10	10	5	5	10	..	..
	79	50	50	44	53	47	54	..
104	50	55	70	60	50	50	60	60
	..	..	..	..	..	45	..	50
	87	62	66	59	59	57	59.3	..

Note: Entries as in Table 1

TABLE 3.—*Data from Ninety-Three Ears with Discrepancies of Ten Decibels or Less Between 500 and 2000 Cps, by Air Conduction*

Ear	250	500	1000	2000	3000	4000	6000	8000
105	—5	—5	5	0	25	5	0	5
	0	0	1	0	2	—2	1.1	..
106	0	0	0	5	0	45	5	5
	..	..	..	..	5	35	..	..
	0	2	9	2	13	0	9.4	..
107	—10	—5	0	0	0	10	0	0
	0	—2	1	—2	1	—2	0.6	..

TABLE 3.—*Data from Ninety-Three Ears with Discrepancies of Ten Decibels or Less Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
108	0	0	5	5	0	30	5	10
	..	..	..	..	5	20	..	..
	0	3	7	4	11	2	8.1	..
109	25	25	30	20	20	30	25	15
	20	20	20	20	20	30	..	..
	0	25	21	23	28	22	25.7	..
110	15	20	20	30	60	80	75	65
	15	20	20	20	50	..	24	..
	0	23	30	22	40	20	36.1	..
111	—5	—5	0	5	10	15	5	0
	..	..	..	..	15	15	..	..
	0	0	4	0	5	—2	5.2	..
112	0	0	5	0	5	10	5	0
	3	2	3	3	4	0	3.5	..
113	—5	—5	5	5	0	10	0	0
	3	2	4	1	4	0	3	..
114	0	—5	0	0	0	0	0	—5
	..	..	..	..	10	5	..	..
	3	—2	0	—1	0	—2	—1.2	..
115	—5	—5	5	—5	35	15	25	—5
	3	—2	4	—3	2	—5	3.5	..
116	0	0	5	10	0	10	50	50
	..	..	5	0	—5	15	..	..
	3	5	6	5	9	2	8.6	..
117	0	0	0	0	5	25	5	5
	..	..	..	..	..	20	..	..
	4	0	3	0	6	0	4.8	..
118	5	0	0	5	5	10	5	25
	..	..	..	0	..	..	..	..
	4	2	3	2	5	0	3.1	..
119	10	5	5	5	20	0	15	0
	4	5	4	4	4	5	4.6	..
120	10	5	5	5	5	5	20	0
	4	5	5	4	5	5	4.9	..
121	0	0	0	0	5	10	20	—5
	..	..	..	5	—5	0	..	..
	5	0	1	0	3	0	3.2	..
122	0	—5	0	0	5	0	5	5
	5	—2	0	—5	0	—2	—0.9	..
123	20	15	20	10	15	20	15	10
	20	15	15	10	10	20	..	..
	5	15	12	13	16	12	15.9	..
124	5	10	5	10	5	10	15	10
	5	10	5	5	5	5	..	..
	5	8	8	7	9	7	8.7	..
125	0	0	0	5	5	15	5	5
	..	..	..	..	..	15	..	..
	5	2	3	2	5	0	4	..
126	5	10	15	20	20	30	20	10
	10	10	10	15	15	20	..	..
	6	15	16	14	19	12	17.4	..
127	0	0	0	0	0	10	10	5
	..	..	..	..	5	5	..	..
	6	0	1	0	8	0	2.5	..
128	0	0	0	0	20	50	45	45
	..	..	..	..	25	..	..	..
	6	0	8	0	15	0	12.1	..

TABLE 3.—*Data from Ninety-Three Ears with Discrepancies of Ten Decibels or Less Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
129	0	0	10	10	20	20	20	5
	6	7	10	6	11	5	9.7	..
130	5	10	10	15	15	20	25	20
	10	10	10	10	15	10	..	..
	7	12	13	11	12	10	13.6	..
131	0	0	10	0	0	35	25	15
	..	..	15	10	10	20	..	..
	8	3	6	2	12	0	10.8	..
132	0	0	10	0	5	30	20	5
	..	..	15	10	15	15	..	..
	8	3	5	2	10	0	9.6	..
133	0	0	5	5	0	5	5	5
	..	..	..	..	0	—5	..	..
	8	3	4	4	4	2	3.6	..
134	—5	0	0	0	0	0	0	—5
	5	0	5	10	5	10	..	..
	8	0	0	0	0	0	0	..
135	—10	0	10	0	5	5	25	5
	8	3	4	1	3	0	5.4	..
136	5	5	0	0	0	30	25	0
	..	..	5	10	5	20	..	..
	8	2	5	1	8	0	8.8	..
137	0	0	0	0	15	80	80	70
	..	..	..	5	10	45	..	..
	9	0	12	0	27	0	19.9	..
138	0	0	0	0	45	50	50	50
	..	..	..	..	40	..	..	..
	9	0	8	0	15	0	12.4	..
139	—5	0	0	0	0	35	50	50
	..	0	0	5	5	15	..	..
	9	0	4	0	11	0	9.7	..
140	0	0	10	10	15	55	45	25
	15	5	15	20	20	45	..	..
	9	7	12	6	20	5	17.8	..
141	—5	0	0	5	0	55	50	25
	..	0	5	5	5	30	..	..
	10	2	10	1	16	0	14.3	..
142	20	20	20	15	15	25	45	35
	5	5	0	0	0	10	..	..
	15	18	19	17	20	17	21.1	..
143	0	0	0	10	45	55	45	45
	..	..	..	10	..	..	..	..
	10	3	14	2	19	0	15	..
144	0	0	0	10	10	25	5	—5
	..	..	..	..	10	10	..	..
	10	3	8	2	9	0	6.8	..
145	0	0	10	10	10	20	5	0
	10	7	10	6	10	5	8.7	..
146	5	5	5	5	5	30	5	—5
	..	..	..	5	10	25	..	..
	10	5	8	5	11	5	9.3	..
147	10	20	20	15	10	30	20	20
	15	20	15	20	20	20	..	..
	10	18	15	17	21	17	20.3	..
148	20	20	25	20	60	80	..	70
	20	20	25	20	..	..	..	..
	10	22	28	22	39	20	37.2	..

TABLE 3.—*Data from Ninety-Three Ears with Discrepancies of Ten Decibels or Less Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
149	5	5	5	5	20	40	50	50
	..	..	..	0	25	50	..	..
	10	5	11	5	20	5	14.2	..
150	5	5	5	5	0	25	30	25
	..	..	..	5	5	20	..	..
	11	5	6	3	11	5	10.1	..
151	5	0	5	10	10	25	20	10
	0	0	5	5	10	30	..	..
	12	5	8	5	11	2	9.1	..
152	0	0	0	10	0	10	40	50
	..	..	5	—5	0	10	..	..
	12	3	5	2	8	0	6.5	..
153	10	5	0	5	20	40	30	15
	..	..	0	15	20	20	..	..
	12	8	6	2	13	2	11.4	..
154	5	15	15	20	10	10	20	5
	5	5	15	20	20	15	..	..
	12	17	12	15	14	15	15.1	..
155	0	0	5	10	10	40	50	15
	0	—5	0	5	10	35	..	..
	12	5	11	5	15	2	13.9	..
156	5	5	5	15	40	50	55	50
	..	..	..	5	40	66	..	..
	12	8	16	7	25	5	18.2	..
157	0	0	0	0	25	40	30	0
	..	..	..	..	15	20	..	..
	12	0	5	0	10	0	9.2	..
158	5	5	10	5	5	5	5	20
	10	5	10	15	10	10	..	..
	18	7	4	6	7	5	6.2	..
159	5	0	0	5	10	10	60	45
	0	0	5	5	10	15	..	..
	13	2	3	2	8	0	6.8	..
160	0	0	10	5	10	35	55	15
	5	5	5	5	15	30	..	..
	14	5	10	5	13	2	12.8	..
161	0	—5	5	0	0	20	55	20
	5	5	0	5	10	25	..	..
	14	0	4	0	7	—2	13.7	..
162	0	0	0	10	15	15	5	—5
	..	..	..	10	20	..	..	..
	14	3	6	2	7	0	4.7	..
163	20	20	20	10	15	10	30	30
	5	5	0	—5	—5	—5	..	..
	10	17	14	13	15	15	16.3	..
164	5	20	30	20	5	15	15	5
	15	15	25	25	15	25	..	..
	15	22	19	19	21	20	21	..
165	10	10	15	10	10	10	10	10
	5	5	5	0	0	0	..	..
	16	12	11	11	11	10	11.1	..
166	15	20	20	15	5	10	40	15
	20	10	10	15	15	15	..	..
	17	18	16	17	16	17	18	..
167	5	0	5	10	10	5	65	50
	0	0	5	10	10	5	..	..
	17	5	6	5	10	2	8.8	..

TABLE 3.—*Data from Ninety-Three Ears with Discrepancies of Ten Decibels or Less Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
168	0	0	5	10	15	20	5	—5
	..	..	5	10	20	25	..	..
	18	5	8	5	9	2	7.3	..
169	15	20	20	15	20	30	25	15
	15	20	15	10	..	25	..	..
	19	18	17	17	21	17	20.6	..
170	5	15	15	20	35	55	15	10
	10	10	15	25	40	45	..	..
	19	17	21	16	28	15	22.8	..
171	0	0	10	10	25	25	30	15
	..	..	10	5	20	25	..	..
	19	7	11	6	12	5	11.3	..
172	35	20	20	20	30	55	65	75
	5	5	5	5	20	30	..	..
..	24	20	25	20	32	20	28.8	..
173	20	20	30	30	30	40	40	40
	20	25	35	35	40	45	..	..
	25	27	28	26	31	25	29.2	..
174	5	20	20	20	20	20	15	15
	10	5	0	0	..	—5	..	..
	26	20	20	18	19	20	19.1	..
175	20	10	15	20	40	40	40	40
	10	0	10	20	45	45	..	..
	26	15	18	15	24	12	29.5	..
176	20	20	20	25	55	55	55	35
	15	15	20	25	45	50	..	..
	26	22	25	22	28	20	29.1	..
177	20	15	5	5	0	10	40	60
	15	10	10	0	..	..	..	..
	26	8	7	6	11	5	10.7	..
178	20	20	20	25	30	35	55	45
	10	5	10	15	25	20	..	..
	28	22	22	22	26	20	25.5	..
179	25	30	30	20	10	20	20	35
	5	5	5	20	25	20	..	..
	28	27	22	23	25	25	24.9	..
180	50	50	55	50	60	60	55	60
	40	30	40	35	40	45	..	..
	30	52	53	52	54	50	52.5	..
181	20	20	20	30	50	65	55	40
	20	20	15	35	40	..	..	..
	32	23	29	22	35	20	32	..
182	40	35	30	30	40	65	65	80
	15	10	10	10	25	35	..	..
	33	32	26	31	42	30	39.3	..
183	20	30	30	20	10	10	10	10
	15	15	15	10	10	5	..	..
	35	27	21	22	21	25	22.4	..
184	40	40	35	30	20	25	20	15
	5	5	0	5	10	10	..	..
	35	35	31	32	31	32	31.7	..
185	40	40	50	50	60	60	65	65
	30	25	35	40	45	45	..	..
	35	47	50	46	52	45	49.3	..
186	30	40	45	40	45	55	70	60
	35	35	40	45	45	55	..	..
	38	42	45	41	45	40	45.2	..

TABLE 3.—*Data from Ninety-Three Ears with Discrepancies of Ten Decibels or Less Between 500 and 2000 Cps, by Air Conduction*—Continued

Ear	250	500	1000	2000	3000	4000	6000	8000
187	25	30	30	40	50	80	75	..
	38	33	41	32	48	30	43.5	..
188	40	45	55	45	50	40	50	35
	30	30	30	25	25	25	..	..
	40	48	45	46	45	45	46.1	..
189	40	40	40	40	30	25	30	25
	20	20	25	25	30	25	..	..
	43	40	35	38	35	40	35.7	..
190	40	40	40	45	50	90	..	..
	10	10	10	30	45	..	..	..
	43	42	49	42	56	40	58.3	..
191	40	40	35	40	20	25	30	15
	0	10	0	10	15	20	..	..
	46	38	36	36	34	37	34.3	..
192	30	40	40	35	40	50	45	25
	5	5	5	10	30	30	..	..
	50	38	32	27	40	37	40.2	..
193	55	50	55	50	35	45	50	75
	30	25	30	30	30	35	..	..
	51	52	49	51	51	50	49.4	..
194	40	45	45	50	65	55	40	30
	55	47	48	46	48	45	44.5	..
195	70	65	70	60	55	60	..	..
	35	35	35	30	35	35	..	..
	60	65	61	63	65	62	65.7	..
196	65	70	80	70	60	75	60	45
	15	20	30	30	30	30	..	..
	73	73	73	72	72	70	71.7	..
197	70	75	75	75	60	65	70	80
	30	35	35	40	50	50	..	..
	77	75	73	74	73	75	71.5	..

Note: Entries as in Table 1.

are self-explanatory. We are now ready on the basis of these results to assess each system of prediction, and to compare them one with another.

The most usual system for predicting one score on the basis of its relation to another score is the standard error of estimate. This datum tells us what percentage of predictions would lie within a specified margin of error. Another technique is the R.M.S. deviation of differences between predictions and observations. However, the measure which appeals with most force to the nonstatistician is simply the average deviation of these differences. If we know which predictive system can take the audiogram and hit the actual speech loss with the least error, on the average, it is likely that further

TABLE 4.—*Average Deviation of Speech Hearing Loss in Decibels for PB Lists Versus Predicted Decibel Loss by Several Systems*

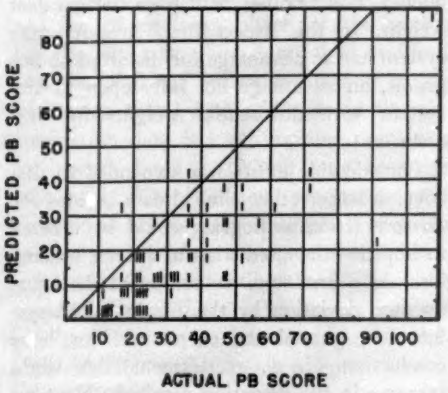
System	Ears			
	Table 1	Table 2	Table 3	All Ears
Multiple Regression	6.0	7.7	5.9	6.3
Two-Worst	6.3 (5.8)	7.1 (6.9)	6.2 (5.3)	6.4 (5.8)
Fowler 1942	6.4 (6.3)	6.6 (6.6)	5.5 (5.3)	6.0 (5.9)
Three-Average	6.6 (5.9)	7.3 (6.8)	6.2 (5.5)	6.6 (5.6)
Fletcher 1953	6.9	7.5	6.5	6.8
Fowler 1941	9.5	...	...	...
Two-Best	13.9 (7.4)	9.6 (6.7)	7.0 (6.2)	9.8 (6.8)
Fletcher 1950	16.8 (9.1)	8.7 (7.3)	6.6 (6.9)	10.3 (7.7)

Note: Figures in parentheses were derived after the original predictions had been corrected with a regression equation.

refinements in statistics will not change our fundamental evaluation among systems.

It will be useful to group our patients according to whether the audiogram is distorted or flat. We selected the same criterion as Fletcher,[12] who grouped those audiograms differing by 20 db. or more between air-conduction losses at 500 and 2000 cps. Our Table 1 is then composed of 64 ears with the same minimum distortion as the 32 ears in Fletcher's Table 2.

For these 64 ears, our Table 4, Column 1, gives the average misestimate by the several systems. The Multiple Regression system succeeds best of all for this group, the Fowler 1942 system next, the Fletcher 1950 last of all.



Prepared from Table 1. Shows extreme underestimation of speech scores for distorted audiograms by the 1950 Fletcher formula.[12]

There were in addition 40 ears with somewhat less distortion but with at least a 15 db. discrepancy between two of the frequencies 500, 1000, and 2000 cps. Table 4, Column 2, shows that for this group the Fowler 1942 system works best, the Two-Worst system next, the Two-Best system last of all.

There remained 93 ears for which no difference larger than 10 db. existed between any two of the frequencies 500, 1000, and 2000. For this group in Table 4, Column 3, of flat audiograms, the Fowler 1942 system worked best, the Multiple Regression and the Two-Worst systems next, the Two-Best system last.

This picture of the Multiple Regression system being superior with distorted audiograms and of the Fowler 1942 with mildly distorted and flat audiograms changes quite a bit when one not only considers the average deviations between raw scores but uses the so-called regression equation to predict. The Figure makes this concept clear. For the 64 distorted audiograms of Table 1, we present a scatter diagram of the relation between speech hearing loss *vs.* predicted loss by the Fletcher 1950 system. It is clear that few of the points fall on the 45 degree diagonal, and, as Table 4 shows, the prediction is off by 16.8 db. on the average. But it is also clear that if the best-fitting line is drawn through the points, as the solid line in the Figure has been drawn, the deviations from this line would be much less than the deviations from the 45 degree diagonal. The equation for this line is as follows:

Predicted speech hearing loss in decibels = 0.98 Fletcher 1950 prediction + 9.2 db.

If one inserts the untreated Fletcher 1950 predictions in this formula for the 64 ears, the new average deviation shrinks to 9.1 db.

Similarly, the other systems can be corrected as follows:

Multiple Regression system:	$X = 1.00Y + 0.30$	(negligible)
Three-Average system:	$X = 0.97Y + 4.4$	
Fowler 1942 system:	$X = 0.94Y + 2.1$	
Fletcher 1950 system:	$X = 0.98Y + 9.2$	
Fletcher 1953 system:	$X = 0.95Y - 0.4$	(negligible)
Two-Best system:	$X = 0.96Y + 9.2$	
Two-Worst system:	$X = 0.88Y + 3.5$	

X = corrected prediction.
Y = previous prediction.

The correction factors in the regression equations given above, ranging up to 9 db., arise from some defect or defects in the way the various systems look at the speech-tone problem.

When the predictions are changed by the regression equation technique, the relative order of the systems changes somewhat, to bring into greater prominence the Three-Average system and one of its shortened forms, the Two-Worst system. For the distorted ears of Table 1, the Three-Average system prediction misestimate shrinks from 6.6 down to 5.0 db. This figure is 1 db. better than the Multiple Regression system. Moreover, even the Two-Worst system, when corrected, yields an average deviation slightly better than that for the Multiple Regression system (though differences of about half a decibel or less are practically and statistically insignificant).

What is the nature of the Three-Average system that it can show appreciably the best prediction for distorted audiograms? Is it because the frequency regions represented by 500, 1000, and 2000 cps are all exactly equal in their contribution to intelligibility? But we know from all other experiments and procedures bearing on the topic that 500 cps is less important by itself than either of the other two regions, and logically should have less than equal weight in a predictive system.

Insight into the power of the Three-Average system can be obtained by considering the success of the Two-Best and the Two-Worst systems.

At first glance the Two-Best does not seem much different than the Three-Average system. It was advanced by Fletcher simply because the Three-Average system often yielded predictions several decibels too high for his group of cases and for the speech sounds he considered. But what it does is to take into account the very great frequency redundancy in speech, and it is the only system which emphasizes this factor. A decade ago we sought for and examined a number of ears with sharply localized dips, up to 25 db., somewhere in the region 1000 to 2000 cps. Dips up to 15 db. were found as low as 1000 cps. For the Harvard Spondee lists these dips had no measurable effect on the speech score. We concluded at the time that because of the many frequencies in the spectrum of any word a principle of equipotentiality existed in the frequency contribution to intelligibility, such that if an ear were defective in one frequency region it could still pick up the word on another frequency channel. Thus, the best heard of the three frequencies must be considered, since it contributes to speech hearing by reason of the frequency redundancy which it can utilize.

Nevertheless, in the present series of 64 ears the Two-Best system does not come off well at all: On the contrary, the Two-Worst system is much more efficient. But note that with this system another principle besides equipotentiality is being emphasized, namely, the distortion of frequency cues. Of the three frequencies, the one which is worst cannot well be ignored, since it tends to contribute to lowering speech hearing by reason of the added distortion it introduces.

Now what the Three-Average system does, in an ingeniously simple manner, is to strike a balance between these two opposing principles, equipotentiality and distortion. In effect, the Three-Average system sets up a series of sliding weights which vary with intensity and with frequency to suit the individual audiogram so that now one frequency, now another, will have the greatest weight. In this aspect the Three-Average system has an advantage for distorted audiograms, an advantage not fully open to any system with differential weights for frequencies.

Considerable effort was expanded to discover whether other clinical data besides the air-conduction audiogram could be utilized to improve the prediction of speech hearing loss. As a first step, we computed the usual average deviation by the Fowler 1942 system but ignored the corrections for bone conduction (i. e., recruitment). A slight increase in the deviation resulted. Next we computed the overestimations and underestimations by the Multiple Regression sys-

tem for those ears from Tables 1 and 2 for which we had no direct measure of recruitment but which had a bone-conduction loss of 5 db. or more at two of the three frequencies 500, 1000, and 2000 cps. The overestimations and the underestimations almost exactly balanced, indicating that to give credit to such an ear for recruitment as shown by bone conduction could only have the effect of increasing the average deviation and therefore be inefficient. Again, on the ears for which we had direct evidence of recruitment, the Multiple Regression system overestimated the speech hearing loss, but by so slight an amount that a correction of only 1 db. would suffice again to balance the misestimations.

We conclude that for our series of 197 ears it does not help to add or subtract values for recruitment.

Our pitch discrimination data were of no direct help in prediction. There were, however, correlationships among three variables: recruitment, pitch discrimination, and the presence of a speech-tone hearing discrepancy. Such discrepancies occasionally appeared with recruitment where pitch discrimination had not deteriorated but seldom if ever were marked where both recruitment and pitch deterioration existed simultaneously. The covariance of these factors has been discussed by us in a previous article.[14]

Consideration of other clinical data, such as type of deafness, shape of the speech intelligibility curve, age, duration of deafness, and the like, has led us reluctantly to the conclusion that by no additions to the air-conduction data can we improve prediction for speech hearing. Others may be more successful. The Pearson product-moment correlation between the Multiple Regression system prediction *vs.* the actual speech hearing loss is 0.90, indicating that 81% of the variance is explained. But we at present conclude that the sources of statistical variance exhibited in the speech reception scores, beyond what can be accounted for by the air-conduction audiogram, have no one or two simple sources and probably reside in the many possible reasons why speech and pure-tone hearing do not always coincide.

These reasons form, indeed, one of the strongest reasons for laboring over the problem of discrepancy. One wishes not to construct a predictive system so that no ear appears discrepant. This is obviously impossible. What is desired is a system which will accurately pick out those ears for which the discrepancy is real, and does not depend upon a juggling of figures.

Since the causes for speech-tone discrepancies are quite different for the two directions of discrepancy, we will treat the data in each direction separately.

First we consider the case of speech hearing better than the audiogram would predict. There were in all 28 cases in which one or more predictive systems assigned a discrepancy of 10 db. or more. On 11 ears there was general agreement. The Multiple Regression system yielded two more with which the other systems did not agree, the Three-Average system yielded two more, and the Fowler 1942 system yielded six more. The Multiple Regression and Fowler systems agreed on four, the Three-Average system dissenting; and the Multiple Regression and the Three-Average systems agreed on three more, the Fowler system dissenting. There were no cases on which the Fowler and the Three-Average systems agreed, with the Multiple Regression system dissenting.

An inspection of the audiograms for those ears which only one system labelled discrepant shows clearly in each case why the discrepancy appeared: The two labelled discrepant by the Multiple Regression system only had precipitate drops at 4 to 6 kc.; the two labelled discrepant by the Three-Average system both had sharply peaked losses at 1000 cps; of the six labelled discrepant by the Fowler 1942 system, five had sharp drops at 2 kc. and one at 4 kc. Thus there will be an occasional audiogram which emphasizes the one weakness in any differential-weighting system; it is difficult to believe that on the basis of one system alone an ear should be labelled discrepant.

The best solution appears to be to label an ear discrepant if the average of the Multiple Regression and Three-Average systems shows a 10 db. or more difference. If this is done, all cases of discrepancy will be correctly labelled except possibly an occasional audiogram with a sharp V at 2000 cps, for which Fowler would predict worse speech hearing than actually occurs.

It is worth noting that the Multiple-Regression-Three-Average method picks out all 10 of those ears for which the pure-tone loss at each of the frequencies 500, 1000, and 2000 cps is 5 db. or more worse than the speech loss. In such a case it is hard to see how pure-tone hearing could subserve the better speech hearing without there being a real discrepancy. The fact that all these ears are correctly identified by the average of the two systems lends some support to the method.

We now consider the case of speech hearing worse than the audiogram would predict. There were in all 27 cases for which one or more predictive systems assigned a discrepancy of 10 db. or more. Of these, all but one would be labelled discrepant by the average of the Multiple Regression and Three-Average systems; that ear showed a loss of 55, 55, and 35 at 500, 1000, and 2000, and the Fowler system credits the 2000 cps value with more sustaining power for speech than is the case, so that for that ear the Fowler system is likely to misestimate.

On all but 1 of these 27 ears, the direction of the speech-tone discrepancy is the same for both the Multiple Regression and the Three-Average system, and a simple average of the two predictions is the best single measure of the amount of discrepancy. If, for example, one relied wholly upon the Three-Average system, one would to some extent mislabel 11 of the 27 ears of this group and 13 of the 28 in the previous group, where speech was better than the audiogram.

Finally, it should be pointed out again that no one predictive system can work as well as some combination of two, whether used for predicting decibel loss for speech or for labelling patients with speech-tone hearing discrepancies. Audiograms come in too bewildering a variety of shapes, and the same shape may have a different significance for speech, depending upon how long the patient has been defective.

Only some combination of systems will tend to cancel out the peculiarities necessarily built into each system. For a variety of reasons already given, we propose a simple average of predictions from the Multiple Regression and Three-Average systems as the most inclusive prediction of speech hearing loss.

One final point, but a very important one, must be made. We have no controlled way of knowing whether a speech score in terms of decibel loss is a linear predictor of actual disability in ordinary living. We think it probably is not; both Fowler and Fletcher have provided curves to pass from decibel loss to percentage loss or compensable loss. On this point the present data do not bear. But a subsidiary question rises whether the PB words are a good sample of the frequency components which make for intelligibility. We conclude that for the present purpose they are nearly ideal. The question is whether the PB lists do fairly sample the phonemes of English as well or better than, say, the Harvard Spondee lists or the Bell Telephone Sentence Intelligibility Lists. From the way the PB lists were constructed, we conclude they do best sample the phonemes of daily discourse.

The question is not whether the exact recordings used, the W-22 records with Mr. Rush Hughes as announcer, cannot be improved or that different announcers might not yield different intelligibility in terms of 0.0002 microbar. That, of course, is true. But the absolute level at threshold is irrelevant here, since we refer to a normal group rather than to a physical reference. Furthermore, there apparently is enough broad peaking in the contribution of various frequency regions to render differences among talkers a negligible quantity, even as between the male and the female voice. In this Laboratory we have in the past gone over much the same ground as reported in this

paper, but using PB recordings made here using four male and four female voices, both spoken and whispered.[16] In view of the fact that the audiogram is only sampled in octaves, and in view of the broad frequency components in all words, it is perhaps not surprising that the relative contributions of frequencies to the intelligibility of these eight voices is not much different at least for the spoken voice. Because of this fact, we used the nationally distributed W-22 records in the present study rather than our own records, reasoning that for our purposes it was irrelevant which were utilized.

We predict on the basis of our experience that while other recordings of other speech materials might show higher face validity for ordinary discourse, nevertheless, the matter of frequency contribution would not differ much from the W-22 records.

SUMMARY AND CONCLUSIONS

The theoretical and practical bases are examined for attempting to predict a hearing loss for speech using only the pure-tone air-conduction audiogram. Previous systems are discussed (1) using the average pure-tone loss at 500, 1000, and 2000 cps, (2) Sabine's system based on the percentage of psychoacoustic discriminations lost, (3) Fowler's 1941 and 1942 systems based on empirical observation, (4) Fletcher's 1950 and 1953 systems based on loudness, and (5) proposals to use loss at the two best heard of the frequencies 500, 1000, and 2000 cps and to use loss at the two worst heard of the three frequencies.

A new system based upon the statistic of the Multiple Regression prediction equation is proposed.

All these systems were tried out on a series of 197 partially defective ears with fairly complete diagnostic information available. Speech used was the phonetically balanced recorded lists' W-22 from the Central Institute of the Deaf. Reasons are given why these lists should serve to investigate the contribution of frequency to the intelligibility of English phonemes.

The results showed that for 64 especially distorted audiograms the Multiple Regression Equation predicted speech scores somewhat better than any other but that for other audiograms the Three-Average system was superior. After an analysis of success and failure achieved by the Two-Worst and Two-Best systems, it was reasoned that the Three-Average system contains within itself two opposing principles: "equivalence," expressing the fact that speech can be understood even with a dip in the audiogram because of broad frequency representation, and "distortion," expressing the fact that a dip in the audiogram introduces some garbling of frequency cues.

It was shown that no one predictive system could be superior for all audiograms, since some audiometric patterns can always be found to exploit the strength or weakness of any one system. It was shown that the most stable system for predicting speech hearing loss was an average of the Multiple Regression Equation prediction and the Three-Average prediction.

It was recommended that the average of these two systems be used in the Navy to assess speech hearing loss.

APPENDIX

The following are notes on the derivation of a Multiple Regression Equation to predict speech reception from the pure-tone audiogram. Find the Pearson product-moment correlations among all the losses at the various frequencies and for speech. Ascertain that none of the relationships depart from linearity. Construct the table of intercorrelations:

	500	1000	2000	3000	4000	6000	8000
Speech	0.742	0.809	0.758	0.518	0.383	0.569	0.486
500	.....	0.851	0.544	0.332	0.206	0.341	0.411
1000	.....	.....	0.602	0.457	0.337	0.418	0.404
2000	.....	.....	.....	0.774	0.537	0.504	0.477
3000	.....	.....	.....	.....	0.837	0.633	0.584
4000	.....	.....	.....	.....	.....	0.744	0.677
6000	.....	.....	.....	.....	.....	.....	0.880

Following the Wherry-Dolittle system, the single most important predictor is 1000 cps. The next most important is 2000 cps; a multiple correlation for these two alone comes to 0.854. Next in order are 4000, 5000, and 6000 cps, raising the Multiple Regression successively to 0.870, 0.884, and 0.894, at about which point diminishing returns make it inefficient to add more variables.

The Multiple Regression of 0.89 means that about 80% of the variance in the speech scores is explained in terms of the audiogram, which is very good as these things go. The problem becomes one of using the partial regression coefficients, found by the Wherry-Dolittle method, to predict the speech score. If hearing losses were expressed in standard scores it would be possible simply to multiply loss at any frequency by its coefficient and add up the results for all five frequencies. Fortunately, it is not necessary to convert the audiogram; one may instead convert the coefficients. Each coefficient is multiplied by the ratio of the mean hearing loss and the standard deviation of that mean. Each coefficient is thus corrected by the data for its frequency. The corrected coefficients are the following:

500	0.252
1000	0.277
2000	0.208
4000	0.181
6000	0.068

These values are used in the final equation. For any audiogram the raw hearing losses at the five frequencies are multiplied each by its respective value, and the sum of the five products is the Multiple Regression prediction of loss for speech.

Using this equation the predicted speech losses for the 197 ears of this study were calculated as shown in Tables 1, 2, and 3. The Standard Error of Estimate for this prediction is 3.2 db., which is interpreted similarly to a standard deviation, namely, that two out of three times the prediction will be within 3.2 db. of the actual speech score.

TABLE 5.—*Table for Computing the Predicted Speech Loss from the Pure-Tone Audiogram Based on the Multiple Regression Equation*

Hearing Loss	Frequency				
	500	1000	2000	4000	6000
5	1.2	1.4	1.0	0.9	0.3
10	2.5	2.8	2.0	1.8	0.7
15	3.8	4.1	3.0	2.7	1.0
20	5.0	5.5	4.0	3.6	1.4
25	6.3	6.9	5.0	4.5	1.7
30	7.6	8.3	6.0	5.4	2.0
35	8.8	9.7	7.0	6.3	2.3
40	10.1	11.1	8.0	7.2	2.7
45	11.3	12.4	9.0	8.1	3.1
50	12.6	13.8	10.1	9.0	3.4
55	13.9	15.2	11.2	9.9	3.7
60	15.1	16.6	12.2	10.9	4.1
65	16.4	18.0	13.2	11.8	4.4
70	17.6	19.4	14.2	12.7	4.8
75	18.9	20.8	15.2	13.6	5.1
80	20.2	22.2	16.2	14.5	5.4
85	....	....	17.2	15.4	...
90	....	....	18.2	16.3	...
95	....	....	19.2	17.2	...

Directions: For each frequency, find the entry in the row representing the hearing loss at that frequency. Sum the five entries.

In order to make the computation easy, a cumulative table (Table 5) has been prepared. To use it, simply find the five entries representing hearing loss and add them up. The sum is the predicted speech loss.

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Pathology and Audiology of Middle-Ear Lesions

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Ten years have passed since the end of World War II, ten years since the first large-scale clinical utilization of penicillin. Fundamental changes in otolaryngology occurred in this decade, changes which seemed menacing to the very existence of otology and which, therefore, lead some otological Cassandras to speak of the "dying profession." More recently the prophets of doom have fewer and fewer followers. On the contrary, a more optimistic outlook seems to prevail. Increasing interest in and enhanced therapeutic activity against hearing losses due to conduction-type defects have become evident. New surgical methods are being developed and new diagnostic criteria are being designed for the purpose of properly selecting and assaying the tympanic pathology.

In the past, the treatment of conduction lesions after the inflammatory phase had burned out was neglected by otologists. Scarification and adhesions in the middle ear appeared to be unalterable, unassailable, and even unpreventible. The interest in the surgical treatment of adhesive nonotosclerotic middle-ear diseases poses many problems. There are not only surgical but also audiological-physiological problems involved.

Our ignorance of simple and fundamental aspects of conduction lesions is embarrassing and hinders the surgical development. These audiological problems of otology are clinical and have to be studied by otologists. It is not to be taken for granted that the audiologist trained chiefly in psychology will be as interested, as prepared, and as helpful in solving these physiological problems as he was in the hearing-aid programs or in helping in the psychological adjustments of patients.

The present and important problem in clinical audiology pertaining to conduction lesions is a systematic comparison between morphological and functional changes. We do not know fundamentally the functional loss, if any, a person will suffer if the eardrum is thickened. What hearing loss is brought about by a calcareous deposit in the pars tensa? What therapeutic effect would be created by removing a calcareous deposit? How much hearing gain can be predicted if a perforation is closed? Are perforations in certain areas of the eardrum more detrimental as to hearing impairment than in other areas? Are there conditions in the middle ear where the sound-conduction system is performing "worse than necessary"? By this is meant that a certain tissue pathology in the middle ear hinders the sound conduction so greatly that its removal, even if accompanied by removal of parts of the sound-conduction system, would improve the hearing. Could, in such cases, a radical mastoid operation be performed in order to improve the function? Even the normal numerous morphological individual differences between temporal bones, in regard to eardrum inclination, individual thickness of the tympanic membrane, window position, etc., are not understood as to their functional significance.

The answer to some of the questions can be given by carefully executed experiments.

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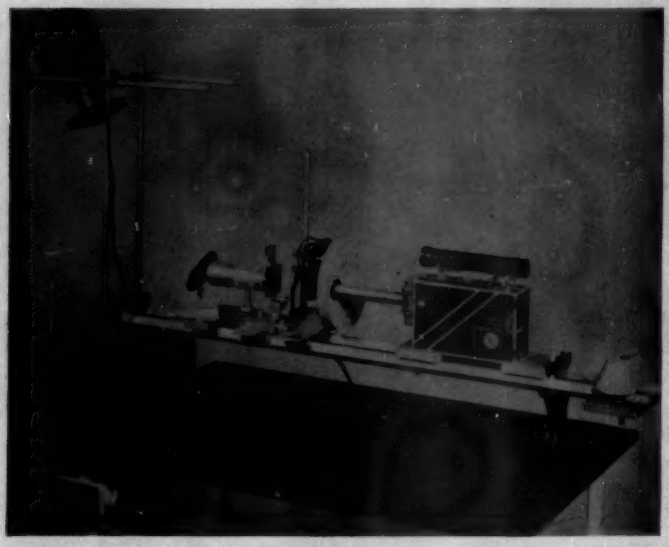

Fig. 1.—The optical arrangement for eardrum photography. The patient is seated with his head fixed in a headrest below the light source. The light source consists of a 2×2 projector with a heat-absorbing glass attached to it. The light shines onto the concave mirror (an ordinary head mirror) while the camera photographs through the hole in the center of the mirror. This gives good coaxial alignment of light and camera axis, which is absolutely essential in photography of as narrow a cavity as the ear canal. Lens, extension bellows, stroboscope (a rotating disc with perforations), and camera make up the rest of the optical equipment.

Nearly all middle-ear lesions can be produced experimentally in animals. The function can be estimated experimentally in certain animals by a variety of methods. It seems to be only a question of planning and of time before the answers for some of the questions can be furnished. Observations on humans can be utilized by objective recordings of the findings. The methods of recording of the eardrum by photographic techniques have been improved recently and made easier and less costly. Eardrums can be photographed readily, and these photographs provide a storage of accurate findings which can be restudied at later dates. Each time, functional tests can be performed simultaneously. Thus, it is possible to make a systematic comparison between morphological and functional changes. Comparisons of this kind have been carried out in a limited way in our own clinical material. Figure 1 shows the set-up for the photographic method. No expensive special camera is necessary. Routine lenses and cameras which are available in all photographic departments of hospitals are utilized. Only a small amount of machine-shop work is necessary to make a lens holder, mirror holder, and camera holder. A smooth-working method for illuminating and focusing of the framing is thus obtained. The photographs obtained with this method were measured quantitatively. The highest object-to-image ratio used in this method is 1 to 5. When the transparencies are projected, a second magnification is obtained by the projection process. The linear projection magnification may be × 50, × 100, or × 200. Pencil sketches were used to outline the enlarged eardrum findings. Perforations, calcareous plaques, areas of fibrosis, and atrophic scars can thus be measured down to fractions of a square millimeter and the transparencies stored for later comparisons with findings obtained at other times. Audiograms are available for each date.

This method seems to be a hopeful approach to sift and to catalogize the vast number of variations of otoscopic findings. It may also be noteworthy to observe the vascular marking and filling of blood vessels and the pulsation of secretion. Naturally, some dynamic phenomena cannot be shown on slides but must be recorded by motion-picture film, for example, the excursions of the eardrum on politzerization or during a Toynbee or Valsalva maneuver. There are advantages to test tubal function by observation of the eardrum rather than by listening through an otoscope. The magnifications used in photographing the human living eardrum or tym-

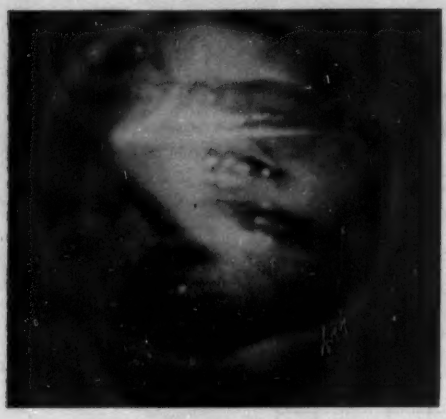

Fig. 2 (Case 1).—Enlarged black-and-white print of a Kodachrome photograph of the eardrum. Pathology characterized by (1) prominence of short process, (2) fold formation posteriorly, (3) Shrapnell's membrane adherent on hammer neck, and (4) several folds and areas of fibrosis in pars tensa intermingled with atrophic areas.

panic cavity are similar to the low magnifications commonly used when examining the middle ear histologically. The living tissues, when enlarged, show many details which are not discernible in clinical otoscopic examination. The term "biomicroscopy of the eardrum"—a term borrowed from the ophthalmologist—is suggested for this method.

There are given in Figures 2 and 3 the otoscopic findings of two patients and the corresponding hearing thresholds for pure tones are presented in Figures 4 and 5. It is evident that the otoscopic picture cannot, at the present status of our knowledge, explain the conduction loss, nor can an otologist "predict," when viewing the otoscopic finding, the functional loss created by the tympanal pathology. It should be our aim to learn to assess each pathological entity and to compose the total picture. As examples, the following cases are presented.

CASE 1.—The patient suffered from a retraction of the eardrum. Figure 2 shows folds, prominence of the short process, and Shrapnell's membrane adherent to the neck of the hammer. Furthermore, the whitish discoloration of the umbo makes one suspect the presence of fluid in the middle ear. In spite of the extensive pathology, the pure-tone threshold was not lowered as much as expected. There was no significant improvement of function after inflation.

CASE 2.—The patient had a middle-ear disease of many years' standing. This foreign-born patient remembered ear suppuration since early childhood. The pathology consisted of a large perforation of the drum membrane in the inferior-posterior quadrant. Through the perforation there was visible the long process of the incus, the stapes head, and a short part of the stapedius tendon. The anterior-superior quadrant showed extensive calcification. The short malleolar process and the handle of the hammer were intact. The chorda tympani was discernible for a short area posteriorly to the short process. The tendon of the stapedius muscle did not show any motion on acoustic stimulation.

The audiograms of these two patients are given in Figures 4 and 5. Both these patients had good bone conduction and are examples of a predominantly conduction lesion. It is apparent that for physiological studies the relatively good hearing of Case 1 is just as significant as the greater hearing loss in Case 2. Furthermore, it seems rather impossible from these two examples to predict the hearing from the otoscopic findings.

CASE 3.—The otoscopic findings of a third patient are given in Figure 6, with his audiogram in Figure

Fig. 3 (Case 2).—A black-and-white enlargement of a Kodachrome photograph of the eardrum. The pathology is characterized by an eardrum defect located in the inferior half and reaching into the posterior-superior quadrant. There the long process of the incus, the head of the stapes, and a short piece of the stapedius tendon can be visualized. The anterior-superior quadrant of the eardrum shows a calcareous deposit. The texture of the eardrum in the posterior-superior quadrant is fibrotic. Short process and hammer handle are intact. Good motility was observed under low-tone stimulation with stroboscopic observation. Rinne (C_{512}) was negative, became positive when perforation was closed by latex membrane.

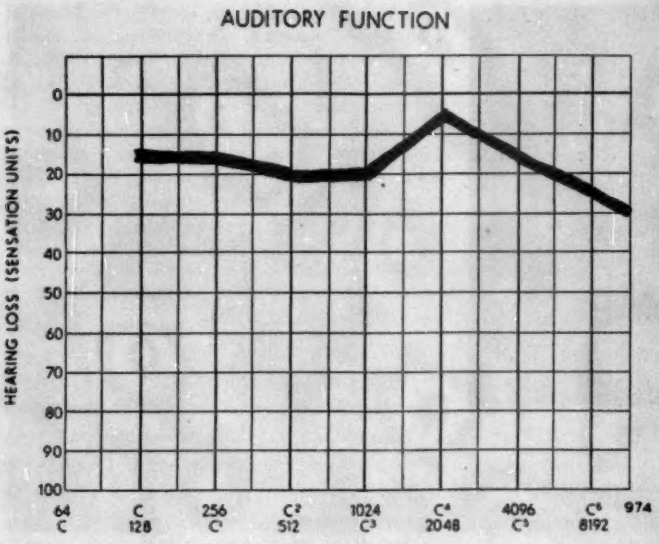

Fig. 4.—The pure-tone threshold audiogram of Case 1. There was good bone conduction. In spite of the extensive visible pathology, the hearing was amazingly good.

7. His findings indicated a mixed conduction nerve type impairment. However, the conductive component was severe and can be treated independently. An analysis of his pathology is as follows:

1. He had, from his perforation, lost some eardrum area. There was less eardrum tissue to collect the sound energy and to conduct it to the hammer handle.

2. He had a calcareous plaque, which most likely reflected some sound energy. The epitympanum was not visible. Other parts of the ossicular chain which were visible, as the incudostapedial articulation, looked normal. Inasmuch as the long process of the incus is frequently diseased, the normal appearance here was significant.

3. Another pathological feature of this middle ear was the direct exposure of the two inner-ear windows to the sound. Normally the oval window receives the maximum of sound energy because the ossicular chain is a far more favorable conductor

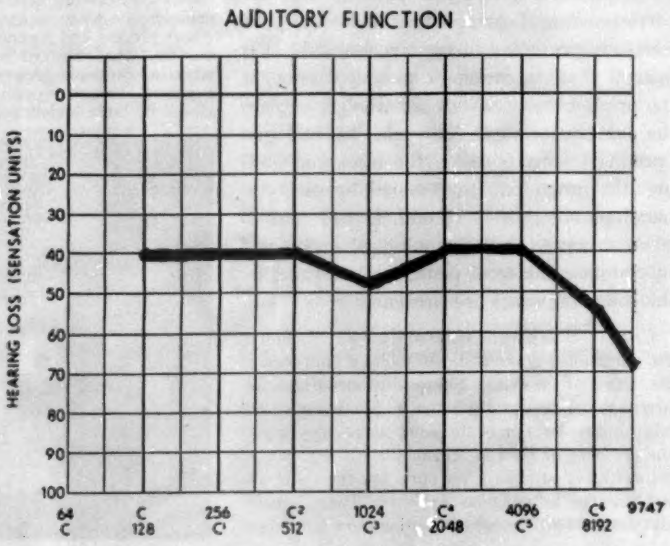

Fig. 5.—The pure-tone threshold audiogram of Case 2. There was good bone conduction.

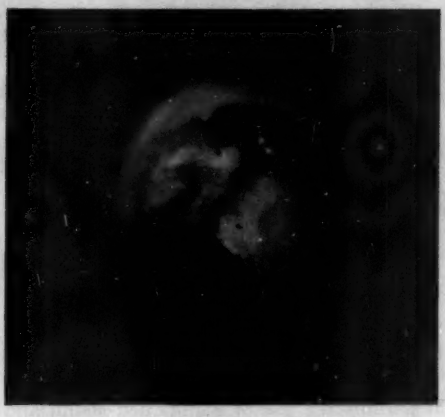

Fig. 6 (Case 3).—A black-and-white copy of a Kodachrome picture of the eardrum of a 37-year-old patient. The pathology is characterized by a large defect in the inferior half of the pars tensa, intensive calcifications in the anterior part of the pars tensa, and less intensive calcification in the posterior half. Short process and hammer handle are standing out clearly.

The eardrum fails to perform its function in several ways. Because of the perforation, a smaller eardrum area collects and conducts the sound energy onto the hammer handle. Secondly, the calcareous plaques reflect sound energy back into the external auditory meatus. Thirdly, the increased stiffness of the remaining drum diminishes the oscillatory response of the vibration system. Fourthly, the air of the middle-ear cavity no longer acts as an encased air bubble, but has an open connection with the outside.

than the air of the tympanic cavity. This is the principle of "preferential sound conduction to the oval window." It was disturbed in this case owing to the diseased ossicular conduction and the open position of both fenestrae.

The tests which can be conducted in the office are as follows: First, a piece of latex rubber is used to close the perforation. The audiological change is noticed. In such an office test a rubber diaphragm is definitely preferred to a piece of moistened cotton; a cotton pellet may touch the stapes and produce a columella effect and/or reach into the round-window niche and produce a sound shadow effect. The otologist working and observing from the outer-ear canal does not control the position of the medial end of the cotton pellet. Therefore the use of a flat rubber membrane is preferred. After the effect of the restoration of the eardum surface has been ascertained, another experiment can be done to find the effect of a fenestral obstruction. Through the eardrum perforation a moist cotton pellet is introduced into the round-window niche, thus restoring the principle of preferential sound conduction.

If, as in Case 3, the audiological result is equally favorable in the two experiments,

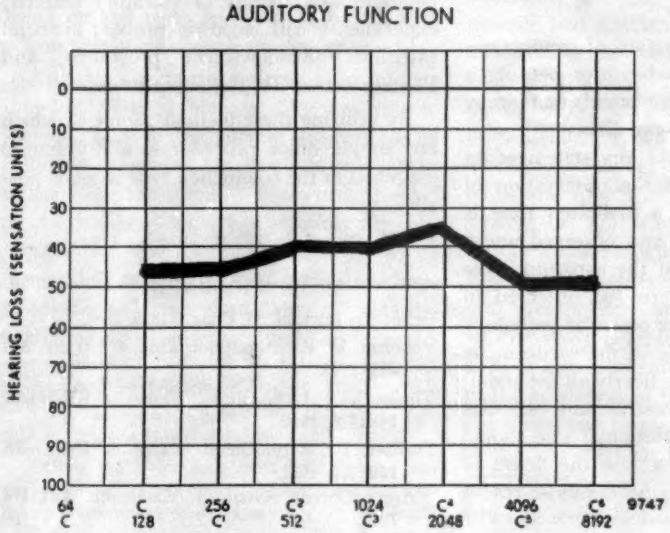

Fig. 7.—The pure-tone threshold audiogram of Case 3. This patient had considerable hearing improvement by application of a round-window prosthesis.

there is a choice of treatment: either an artificial eardrum or a tympanal prosthesis in the round-window niche. In making the decision which treatment to use, the amount and frequency of tympanal discharge should be taken into consideration. Case 3 had a dry middle ear. Therefore, a latex rubber membrane was fitted. In my experience, the hearing improvement is more easily obtained and longer maintained by an artificial eardrum than by a window prosthesis. If, however, the ear were moist, the artificial eardrum would be contraindicated and the ointment prosthesis in the round-window niche would be the method of choice.

COMMENT

In all cases a minute survey of the eardrum pathology is suggested. Photographic recording of eardrum pathology and storage of findings are advantageous. Quantitative measurements are possible and comparable with later findings.

The otoscopic and functional findings of sound-conduction lesions can be analyzed and subdivided into four subentities of sound-conduction pathology.

1. Diseases of the air-eardrum boundary (drum pathology)
2. Diseases affecting the sensitivity of the ossicular chain (ossicular pathology)
3. Fenestral pathology
4. Tubotympanal pathology

The careful differentiation of conduction-type lesions into the subentities permits a better choice of surgical or prosthetic therapy in patients with middle-ear disease.

It is too early to make any statement on the diagnostic value of the observation of vibratory patterns. If a low-pitch tone is directed into the canal and observed under stroboscopic light and if the eardrum gives a good oscillatory pattern (as observed in Case 2), then perhaps an ossicular pathology is excluded. If covering of the eardrum by latex membrane restores nearly all the audiological loss, then a fenestral and ossicular pathology at least of substantial size should be ruled out. In such a case the defect of eardrum area seemed to be the most potent pathological entity.

The clinical diagnosis of stapes fixation can be studied by the quantitative Gellé. Using, as Zoellner and Thullen have shown, an audiometric sound source (in contradistinction to a tuning fork, as used in the classical Gellé) the attenuation in the Gellé position can be measured and expressed in decibels.

Acoustic probes have a place in the office work-up of a deafened patient. Pohlman used a cardboard with a probe, while Zoellner utilizes a receiver with a probe attached to it. The electronic device of Zoellner naturally is superior to Pohlman's probe because it permits the use of maintained sound and measurable differences.

SUMMARY

Postinflammatory conduction lesions seen after the inflammatory phase has burnt out have long been neglected by otologists. In planning a therapeutic approach it must be realized that nonotosclerotic impedance deafness is varied by location, by degree, and by physiological significance. In comparison, the pathology of clinical otosclerosis is monotonous, as it is always located in the stapes area. Nonotosclerotic lesions should be analyzed as to their significant pathology.

The methods are the following: Eardrum photographs; a study of vibratory pattern; experiments with acoustic probes; artificial eardrums; window-niche prosthesis, and Politzer maneuver, Gellé maneuver.

By utilizing these methods, some of which are simple office procedures, a differential diagnosis of the conduction type is made possible.

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Speech and Voice Training of the Deaf

Report on Some Techniques and Experiments

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A HISTORICAL INTRODUCTION

It was no less a man than Aristotle who with his pronouncement that those who are born deaf are also senseless and incapable of reason [1] did a tremendous and lasting disservice to the deaf. Aristotle also implied that the deaf could not possibly be taught to speak because their tongues were tied. For the next 2000 years, Christendom was to "live and die in Aristotle's work,"[2] and his science, or at least a smattering of it, became known over the whole civilized world. Thus, no educated man would waste his time and energy in attempting to educate the deaf, an impossibility acknowledged by all authorities. Incapable of instruction,* uncomprehending and incomprehensible, they were regarded as being on the same level as the brutes with whom they were classed for centuries.

Aristotle's authority decreed even the terminology of the language. One has only to look at some of the current expressions in different languages. In English, one speaks of the "deaf and dumb." The word "dumb," an Anglo-Saxon term, has certainly the connotation of the German "dumm," which means dull or stupid. In German one says "taub-stumm"; the word "stumm" also derives from the older form "dumb" or "dumpf." The French call it "sourd-muet"; the word "muet" has its etymological stem in the Latin term "mutus," meaning dumb. If in modern language the term "mute" means without speech, it carries in its wake the implication of "dumb."

Aristotle's prestige made universal the belief that the deaf were beyond human help and could never acquire speech. St. Augustine, as we might expect, in the "De Quantitate animae," shared Aristotle's ideas about the nature of dumbness. Thus medieval thought was satisfied that the deaf could not be taught and saw no point in essaying the impossible. Any other notion would have seemed absurd. So strong was Aristotle's impact on science that when, in 1550, the first attempts were made to educate the deaf written communication was taught, without the intervention of oral speech. The dam that blocked the way to educating the deaf was eventually pierced, and then only was it realized that instruction was the only means to overcome the mental deficiency and ignorance of the deaf. It so happened that the entire history of education of the deaf has been divided by two trends of thought: (1) whether one should concentrate on the method of oral speech in addition to reading and writing or (2) whether the instruction of sign or gesture language should be preferred. Although the contemporary trend is to prefer oral speech, there are a number of reasons which make a definite and final decision difficult. A historical survey of the developments in the education of the deaf will explain the circumstances responsible for the evolution of the two methods.†

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From the Speech Clinic of the Manhattan Eye, Ear and Throat Hospital.

* Pliny the Elder and Galen just repeated and supported the current belief that dumbness is a necessary concomitant of deafness.

† With permission of the author, some of the following historical data are taken from Fröschels.[3]

During the 16th century, the Italian doctor and mathematician Girolamo Cardano [4] attempted to teach the deaf and dumb to write and thus to acquire the opportunity to express their thoughts by means of language. In his footsteps followed the Benedictine monk Pedro de Ponce ‡ (1570), Bonnet [6] (1620), and de Carrion § (1650) in Spain. Rev. John Wallis [7] (1653), John Bulwers (1648), and Watson [8] introduced those methods in England. The Italian L. Ferreri [9] gave us a modern Italian translation of the very interesting treatises written in the 17th century by von Holden and Volta. In 1642, in Germany, Dr. Johan Camerius published a pamphlet in which he insisted that the deaf and dumb be given the opportunity of an education. He too suggested that instruction in reading and writing be given the most prominent place.

Johan Konrad Ammans,‖ a Swiss physician, was the first to be successful in teaching the deaf and dumb articulated speech. He instructed them simultaneously in lipreading.¶ At the end of the 17th century he put his extensive experience down in two books, which for many years were considered the outstanding publications in the field. In this work he also gives therapeutic advice for other kinds of speech defects.

Ammans' work was continued by George Raphael,[12] whose "improved methods" were published in 1718.

Lischwitz,[13] a famous physician and professor of medicine in Kiel, also used Ammans' method and published his experiences in a work that appeared in 1719.

Jacob Rodriguez Pereira [14] in 1744 presented a memorandum to the French Academy of Sciences in which he recommended the public use of his method, which combined oral speech and the use of sign language and writing. His pupils Ernaud and Deschamp were his successors after his death.

Eventually Samuel Heinicke [15] succeeded in awakening enough interest in the German government so that means were provided for the purpose of erecting the first school for the deaf and dumb. It was founded in Leipzig in 1778 and was very sucessful. Heinicke intentionally avoided the use of sign language and taught his pupils only oral speech. His advice for the instruction of the deaf was to concentrate on oral speech, besides reading and writing, and to abstain completely from instruction in sign language. When the methods are combined, sign language always becomes predominant. Heinicke's method is also known as the German method.

In contrast to the German is the French method, which was devised by the famous Abbé Charles M. de l'Epée. His curriculum,[16] based on Pereira's writings, included sign language and writing but neglected articulated speech. Epée was the founder of the first institute for the deaf, in Paris in 1760. This institute was soon taken over by the French government. Epée died in 1789, and the Abbé Sicard became director of the institute, a post which he retained until his death, in 1822.

Epée and Heinicke exchanged vivid arguments concerning the mutual advantages of their methods. Even today the writings of these two men, their opinions and suggestions, are of great interest to the educator of the deaf.

In the 19th century the education of the deaf advanced by great strides and many teachers of the deaf gained great reputations and fame.

In the United States the first school for the deaf was established in the second decade of the 19th century.[17] The seat of this first permanent school was Hartford, Conn. Thomas Hopkins Gallaudet, a young theological student, was chosen as instructor.

Best relates that when Gallaudet went to England to study the techniques employed

‡ de Ponce, P.: Description of his method in Vallerius.[5]

§ de Carrion, E. R.: Left no publications.

‖ References 10 and 11.

¶ The term "lipreading" has become obsolete. It has been recently replaced by the term "speechreading." However, in my opinion, neither term explains the process. The deaf does not "read" at this occasion but has learned to understand oral speech by means of vision. Such an activity or such a skill may best be described by "visual speech perception."

for the instruction of the deaf he found a monopoly and discovered that the schools in London and Edinburgh were closed to him, the very schools where the oral method was taught. He traveled to Paris and was warmly welcomed by the Abbé Sicard. "But for this fortuitous event," Best writes, "the whole process of instruction of the deaf in the United States might have been changed."

After a year of close observation, Gallaudet returned to America, bringing with him one of the teachers from the Paris institute, by the name of Laurent Clerc. A charter was granted to the society in 1816 by the legislature of Connecticut, and a sum of $5000 was appropriated for the benefit of the school. The school was inaugurated on April 17, 1817, under the name of The American Asylum at Hartford for the Education and Instruction of the Deaf and Dumb. Later the name was changed to The American School for the Deaf. Gallaudet remained at the American Asylum until 1828, when he retired because of impaired health. Best # describes him as a man of rare qualities, who "brought his task a high conception of duty and a complete forgetfulness of self, together with a singular beauty of character and a great human love, that could have existed in few other men."

Soon after Hartford, New York and Pennsylvania followed and established schools for the deaf. In New York it was Rev. John Stanford and in Philadelphia David Seixas who became the organizers of these first schools. It was Hartford, New York, and Philadelphia where in the United States the pioneer schools for the deaf were built.

However, as time went on, the possibility of teaching oral speech to the deaf was taken more and more into consideration. Attention was focused on Germany as the home of the oral method. In 1843 Horace Mann, the Massachusetts educator, and Samuel Gridly Howe * traveled to this country and made on their return favorable reports on the results of oral education, results they were able to observe in Germany. After these reports the movement for oral instruction for the deaf was started in the United States.

In spite of the fact that approximately one-third of all deaf children in the United States are taught in a strictly oral environment without any contact with sign language, among the adult deaf the sign language shows no indication of disuse or abatement. The deaf themselves consider the sign language as their mother tongue; they employ it in conversation with one another, in their business and social meetings, and in their general gatherings.

According to Best, they regard it as one of the few joys remaining to them. When criticism of sign language became stronger and its challenge more peremptory, the deaf even went on the offensive because they considered themselves as the only ones in the position "to judge the matter that is so vital to them"†; they protested that speech acquired without the sense of hearing may sound harsh, unnatural, disagreeable, and difficult to understand; it is also almost inevitable that many words would be mispronounced.

Formally and informally, writes Best, the deaf have insisted upon retention of the sign language. In their convention they speak of the sign language as "a most beautiful language of priceless value to the deaf" and object to any movements to "impair or destroy or restrict it."

In 1938 and 1940 the Empire State Association of the Deaf took the following resolution:

> The educated deaf bear overwhelming witness to the truth that the sign language and manual alphabet are the most practical, convenient and dependable medium of expression for those without hearing.
>
> This Association reaffirms its historical allegiance to and support of the graceful sign language and manual alphabet, and commends all efforts made for their preservation and extension to the end that they may be passed on as a precious heritage to enlighten and inspire coming generations of deaf.

Best,[17] p. 393.

* S. G. Howe was principal of the Perkins Institution for the Blind in Boston and concerned with various philanthropic undertakings.

† Best,[17] p. 531.

Fröschels ‡ has investigated very carefully whether instruction in oral speech or in sign language is to be preferred for the deaf. He concedes that for the congenital deaf lacking any functional hearing § the sign or gesture language is the most natural one. It is also used by a normal person when he communicates with a stranger who does not understand his language; it is used whenever one wants to avoid noise. What heights of accomplishment can be reached by sign language are mentioned by Kussmaul,[18] who once saw a society of deaf people perform a tragedy of Shakespeare using sign language only. He adds that the proof is still wanting as to whether sign language is not better adapted than speech to express the perfection of a Shakespearean play.

In another author's [19] opinion, the importance of pantomimic signs and gesture language should not be underrated. There is no doubt that it has its deficiencies; however, no one can tell how it would have developed if, as it is the case with oral language, the joint effort of hundreds of generations had contributed to perfect it. The question whether human reason would have developed in the same manner if gesture and sign language had predominated is difficult to answer.

Fröschels ‖ concludes that

> for the deaf, sign language is the natural substitute for the missing oral language and should not a priori be rejected in those cases where articulated speech has to be created artificially. Yet, many important reasons speak for the instruction in oral speech and against the use of sign language. In the first place, sign language, when expertly used, becomes a secret language limited to those who learned it. The deaf who has been only instructed in sign language does not acquire "lip reading" so that he is unable to communicate with anybody beyond his narrow circle; confined to a small community, he stays isolated from the rest of the world.

The other objection made by Fröschels is that sign language can be seen at a great distance, so that everybody who knows it is able to "listen."

In order that sign language may become adjustable to expressing complicated thought processes, the finger alphabet was invented ¶ and taught in all institutions for the deaf. It is, however, a known fact that the deaf prefer to use the sign or gesture language, since one single gesture signifies a whole word, whereas communication with the finger alphabet requires as many signs as a word has letters. A deaf person confines himself in most cases to spelling only names and to completing some signs for clarification.

Although Fröschels is convinced that gesture language in its visual form and picture character conforms better with the mentality of the deaf, he advocates instruction in oral language.

An author who raises serious charges against sign or gesture language is Best.# He too takes exception to the fact that it makes the deaf a class apart, builds up among them a feeling of clannishness, and separates them by a wide, unbridgeable gulf from the general population. According to Best, it creates a powerful obstacle to the learning of any proper language, whether it is written or in its spoken form. In addition, wherever the sign language is permitted, or even merely tolerated, oral speech cannot make any substantial headway, because the deaf will immediately relapse into the former, and into "mutism." Sign language is often accompanied by distortion of the face or uncouth facial movements, which frequently provoke a revulsion of feeling among those who do not understand the language. Even if oral language and lipreading (visual speech perception) are not perfectly mastered by the deaf and although it leaves them still deaf, it should at least not leave them "dumb."

The system which favors the oral method combined with signs is known as the "American combined system." At present the slogan is propagated that the most important problem is not so much speech but better speech.

‡ Fröschels,[8] p. 124.

§ Interpretation of the term is given in the following pages.

‖ Fröschels,[8] p. 124.

¶ There are two systems of finger or hand alphabets: One is limited to the use of one hand; the other makes use of both hands.

Best,[17] pp. 524-526.

All educators of the deaf have become aware that the speech and language patterns achieved determine the social status, educational goals, and psychological adjustment of the acoustically disabled. Intelligibility, intelligent speech, fluent written language, etc., become indications of the deaf's intelligence and social adjustment.

HOW TO OBTAIN "BETTER" SPEECH FOR THE DEAF

Although more than 200 years have elapsed since it has been demonstrated to the world that the deaf are not necessarily "dumb" and can be taught to speak, the old attitude, inherited from antiquity, retained some of its power and the habit of speaking of the deaf and dumb persisted. Not until 1930 was a terminology worked out and exact distinctions between the deaf and the hard of hearing were defined.

The Committee on the Deaf and Hard of Hearing of the White House Conference in 1930 set up the following definitions:

> The "deaf" are those who were born either totally deaf or sufficiently deaf to prevent the establishment of speech and natural language; those who became deaf in childhood before language and speech were established; or those who became deaf so soon after the natural establishment of speech and language that it has been practically lost to them.
>
> The "hard of hearing" are those who established speech and ability to understand speech and language, and subsequently developed impairment of hearing. These children are sound conscious and have a normal, or almost normal, attitude towards the world of sound in which they live.

In 1937, a Conference of Executives of American Schools for the Deaf adopted definitions in purely auditory terms:

> The deaf: those in whom the sense of hearing is non-functional for the ordinary purposes of life. This general group is made up of two distinct classes (*a*) the congenitally deaf—those who were born deaf; (*b*) the adventitiously deaf—those who were born with normal hearing but in whom the sense of hearing became non-functional later through illness or accident.
>
> The hard of hearing: those in whom the sense of hearing, although defective, is functional with or without hearing aid.

The first who drew attention to the phenomenon that many of the so-called congenital deaf do possess some residual hearing was Dr. E. M. Itard * at the beginning of the 19th century. He attempted to make use of this residual hearing for speech improvement by means of exercises. Itard, however, proceeded on the wrong assumption that such residual hearing could be increased and developed by means of exercises. A systematic study as to the residual hearing of the deaf was made by V. Urbantschitsch [21] in the 90's of the last century. It was he who discovered that frequently only the hearing for high and low tones was missing. Urbantschitsch recommended hearing exercises for all those deaf children who, after a period of practicing, obtain a hearing sensation.† If no hearing sensation can be obtained, hearing exercises have to be discontinued. For his exercises Urbantschitsch recommends practicing with musical tones on different pitches and stimulation with various vowels. He suggests that words be introduced very soon, showing the object or a picture of the object the word designates, in order to obtain correct object associations. The showing of pictures or of objects makes the hearing exercises more interesting and enjoyable. The intensity of the voice used should never be raised above normal conversational level. Urbantschitsch limits the exercises to half an hour daily and advises that the pupil practice by speaking into his own ear by means of the tube.

I introduced in 1942 [22] a simple improvement of this technique, which required only a minimum of equipment. I constructed an apparatus with two glass larynx bells, both connected by means of a Y-shaped olive and two rubber tubes to the ear of the patient.

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* Dr. E. M. Itard [20] gained considerable renown as an educator of Victor, known as "the Savage of Aveyron."

† A prolonged vowel sound is spoken directly into the ear of the deaf by means of a larynx glass bell. The larynx bell is connected with a rubber tube measuring approximately 2 ft. to a glass olive. The olive has to be inserted into the external auditory meatus of the deaf person.

One larynx bell is to be used by the instructor, the other by the patient. The two larynx bells enabled the patient to repeat immediately the exercises presented by the instructor and thus to compare the latter's speech patterns with his own.

In the age of electronics, I feel as if I were describing some tools of the stone age. Yet one has to remember that the double-belled Urbantschitsch hearing tube seemed a short 15 years ago—in European countries at least—a very efficacious device. It was cheap, easy to handle, bringing speech and voice to the ear with a minimum of distortion, and if, by chance, one of the glass bells broke, one did not need an electrical engineer to repair it. The most valid objection to it was that a normally hearing child is from the very moment of his birth bathed in a world of sound, whereas the deaf child has in comparison only limited auditory stimulation, even with the opportunity of receiving hearing exercises a few times a day.

Recent advances in the physics of sound made improved methods possible. After introduction of the radio, Fröschels suggested headphones be made available to deaf children, so that they could listen to music by radio.

Eventually, some 37 years ago, the invention of the vacuum tube brought the possibility of speech amplification for the deaf into easy reach.

Dr. Kerridge,[23] in 1935, made an investigation in the London County School for the Deaf and Partially Deaf. He expressed the opinion that 84% of the pupils of that school might be able to benefit from amplified speech.

According to studies made at the National Institute in Paris [24] and at the Clarke School for the Deaf in the United States, the percentage of pupils who have any usable hearing over the speech-sound range approximates 45 to 50. Dr. Ruth Guilder [25] stated: "The remaining 50 to 55 per cent, who cannot be helped through hearing in the actual discrimination of speech sounds, may be helped toward more normal accent inflection and phrasing through amplification by bone conduction, thereby acquiring a more normal rhythm of speech." For those who have some residual hearing over the speech range, the vacuum-tube hearing aid makes better hearing no longer a dream but a reality.

Guilder [26] is convinced that the majority of deaf children can be given better hearing and, through better hearing, can acquire better speech and better language and become happier, better-adjusted persons. In order to attain this goal, she advocates the following measures: Whenever a hearing defect is detected sufficient to interfere with the hearing of speech, "lipreading" should be the first remedial therapy to be suggested. It should, however, always go hand in hand with the greatest possible use of hearing, so that a hearing aid in addition to lipreading becomes the best remedy, when the unaided ear can no longer easily discriminate certain sounds. Guilder, as a result of her studies, believes that "it has been proved conclusively that sight and hearing together give the highest percentage of speech intelligibility at every age level."

On this basis Guilder has worked out a clinical program [26] adjusted to the remedial problems of the hard of hearing and partially deaf child. This program includes individual instruction in speech correction, lipreading, and listening exercises with the benefit of hearing aids. When auditory handicapped children are reared with the help of this program, the result may be such that a considerable number may be able to live in their own homes and go to regular classes. Transferring the children to special boarding schools should be avoided because it always involves considerable expense to the State or to the parents. Guilder strongly advocates the start of rehabilitation at the age of 3, in order to utilize more of the precious years of early childhood, which are the years of greatest speech readiness and, if lost, can never be completely regained. The second, third, and fourth years of a child have been provided by nature in which to acquire speech and language. At 6, the child should be ready to

grasp new material rapidly by means of hearing.‡

Very recently, Clarence V. Hudgins made a study as to the usefulness and effectiveness of auditory training for profoundly deaf children, who have hearing losses in the speech range of 80 db. and more. So far in the schools for the deaf only partially deaf children with hearing losses ranging from 60 to 70 db. have been equipped with hearing aids, while, for all practical purposes, hearing aids have been considered as being useless for the profoundly deaf children. The evaluation of Hudgins' experiment indicates that even profoundly deaf children benefit considerably by the constant use of a powerful high-fidelity group hearing aid. Hudgins [28] states that "improvements in speech perception, general educational achievement, and to a lesser degree speech intelligibility were demonstrated." All those pupils who had the advantage of the powerful hearing aid showed much greater gains than those pupils who were trained by means of the hearing aid which was used as standard equipment throughout the school. The author accounts for the failure in improving the speech intelligibility by the age level of these pupils (the ages ranged from 9 years 10 months to 12 years 10 months) and believes that a group of younger pupils would manifest increased improvement in speech intelligibility following auditory speech stimulation training. He later selected another group, with the age range of 8 years 11 months to 10 years and 6 months. The findings of the second experiment carried out with the younger children during a two-year period evidenced, as predicted, that the gains in speech intelligibility for this younger group, with one exception, were significantly higher than those of the control groups.

As a consequence of his experiments, Hudgins advocates the availability of high-powered hearing equipment for profoundly deaf children. Such equipment should be "capable of presenting undistorted speech signals at levels up near the threshold of pain, and with a power-limiting device that protects the ear against discomfort or acoustic trauma."§

In spite of Hudgins' conclusions, other educators of the deaf express different opinions. Powrie Vaux Doctor,[29] for example, writes: "The hearing aid is without doubt one of the greatest achievements in the field of hearing in this century. However, the fact that many hard of hearing people do benefit from hearing aids has led to the mistaken belief that hearing aids can benefit all deaf people. Of the 21,545 pupils reported as being in schools and classes for the deaf during the year 1952-1953, 6,883 were reported as using a group hearing aid and 4,552 as using an individual hearing aid." Thus, according to these data, 10,110 children in the United States cannot benefit from hearing aids.

It is not in the scope of this article to review the tremendous amount ‖ of publications written on auditory training of the deaf and the hard of hearing. However, two trends may be detected in most of these studies: first, the recommendation of increasing the hearing acuity by means of better and more powerful machinery,[30] and second, the advice of training the hard of hearing person to make more effective use of his hearing remnants by means of instruction for better discrimination of the speech sounds, those sounds or words his hearing can only distinguish very vaguely or not at all. The latter can be done with or without hearing aids.¶

‡ Guilder suggests that the remedial program be carefully adjusted to the age of the child. For preschool-aged children it should take place in a nursery school setting, with play material and with an observation and play-conditioning technique. Guilder recommends that the hearing aid be first introduced as a play telephone, and a word-picture game used for measurement of his understanding of language and for his ability to hear speech. For the education of older children, I have been using very successfully the manual "Auditory Training." [31]

§ Hudgins,[28] p. 287.

‖ See the *American Annals of the Deaf*, founded at the American School for the Deaf in Hartford, Conn., in 1847 and the Volta Library in Washington, D. C.

¶ References 31 and 32.

EXPERIMENTS BY MEANS OF TOUCH IMPRESSIONS

Teaching speech to a child born deaf is without doubt the most difficult job in the educational world. As already previously mentioned, one of the methods to teach speech to the deaf is to substitute for the sense of hearing vision and touch. The deaf child, during his speech lesson, puts one of his hands on the cheek or beneath the chin of his teacher, his other hand on his own throat and attempts to imitate what he sees and what he feels. Experiments to improve this rather primitive method were made by R. H. Gault,[33] who successfully communicated vocal vibrations instrumentally to the fingers or palms of deaf persons, so that the spoken word could be felt by a considerable group of deaf persons simultaneously. Gault attempted to train his pupils to understand speech through the medium of touch impressions. In addition, he worked out methods to aid the deaf to improve their vocal expressions. The deaf person, by feeling his own voice and that of his teacher, receives some cues to guide him in the act of speaking; by this means the deaf may receive sound and learn to interpret it and to control their own vocal expression. Gault claims that some of his pupils learned to interpret and to pick up words and isolated sentences. Two of them became so proficient that they were able to interpret an entire story by its feel communicated through the receiver of Gault's apparatus to their fingers.

Gault observed that not only the direct enunciation of speech sounds has been improved but also what he calls accent, emphasis, rhythm, and tempo.

It was by pure chance, without any knowledge of Gault's previous experiments, that I thought along similar lines. In my profession of speech and voice pathologist, I have been frequently faced with the problem of improving the voices of my patients, although patients whose voices need improvement do not necessarily have to be deaf. It happens, however, frequently enough that such patients are not endowed with the capacity to produce a correct musical tone or to imitate one [34] when they hear it. Such persons are usually called tone deaf. If ever such a tone-deaf person has, for one reason or the other, to improve his voice, the task of the voice therapist becomes a very difficult one. On such occasions I prefer to use electrically driven tuning forks calibrated to a certain frequency which, when brought in contact with the patient's thyroid cartilage, stimulate his vocal cords to produce vibrations in unison with the tuning fork.

At my work at the Veterans Administration Mental Hygiene Clinic I recently had to rehabilitate the voice of a patient who had undergone extensive laryngeal surgery. After recondition therapy of the breathing mechanism, the patient eventually learned to speak with normal loudness; however, the pitch of his voice was one octave higher than before surgical intervention. (Before he was operated on, the pitch of his speaking voice ranged from A to e; the pitch the patient could produce after surgery was around a [or 217.50 d.v.], the pitch of a woman's speaking voice.) In spite of practice, which included all the exercises recommended for achieving lower pitch, such as chewing and jaw rocking, no baritone voice could be obtained. At last, I remembered that my patient was a draftsman at the Bell Laboratory in Summit, N. J. I inquired whether he could devise an apparatus which when brought in contact with a healthy normal male larynx would pick up the vibrations of the vocal cords of this larynx during phonation and transmit these vibrations "amplified" to the larynx of the patient. These amplified vibrations were thought to provide the patient with a stimulus needed for the production of a normal-pitched male voice.

After a few trials, the patient, Mr. R. Nielsen, succeeded in constructing a suitable apparatus, by means of which I eventually accomplished my goal and obtained in a relatively short period of time the correct pitch of the patient's voice.

Mr. Nielsen describes the applicability of the machine he constructed with the following words:

> The method of using this apparatus comprises the steps of applying the amplified vibrations of the instructor's voice to the neck of the patient in the region of the larynx through suitable transducers, then having the patient repeat the sound into a microphone; thus the patient's own sounds are amplified and applied to his own larynx by means of the same transducers. It is by comparing his own sound vibrations with those of the therapist, that the patient may determine whether the sound he produces is identical with the one produced by his instructor. Those transducers may also be used to determine whether the pitch of the patient's tones remain constant since any variation in pitch will directly influen e the number of frequencies in the transducers.

When Mr. Nielsen's cure had been completed, he was kind enough to let me have his device, and since my activities include the task of improving the speech of the profoundly deaf, the ownership of the apparatus enabled me to put into reality an old dream of mine. I put it to use while working with the profoundly deaf.

The two main problems while teaching speech to the deaf are (1) to obtain a good voice and (2) to obtain the correct enunciation of the speech sounds. However, every experienced teacher of speech to the deaf is aware that lack of rhythm and melody manifest in the speech of the deaf proves to be a very disturbing deficiency.

To teach and to improve articulation of the speech sounds, we had to rely on the well-known classical methods of teaching speech to the deaf. However, Nielsen's machine, which we called "the voice stimulator," became of unique value to improve rhythm and melody.

The deaf person does not know what to do with his vocal organ, and that is the reason why the voice of such a person sounds harsh, forced, and disagreeable. His ear cannot guide him to imitate the sounds of his environment. When his throat is brought into contact with the just-described larynx transducers carrying the amplified vibrations from the instructor's vocal cords to the deaf's larynx, the vocal cords of the deaf person receive the stimulus to assume vibrations in unison with the instructor's larynx. It was an easy means to elicit well-produced tones on a variety of pitches. However, the application of these tone modulations for spontaneous speech patterns takes a much longer period, and the tone modulations are easily forgotten if they are not practiced again and again with the help of the vocal stimulator.

Those who have worked with the deaf know how difficult it is to make the deaf understand about accent and what syllable should be stressed in a polysyllable word. Before we had the vocal stimulator we used to beat the rhythm of a word into the palm of our deaf pupil. For example, "Saturday" was one long beat and two short ones. Through the vocal stimulator, the stressing of the first syllable, or any other syllable, can be exactly felt on the larynx, since it has its direct effect on the larynx transducers. Thus it is easy for the deaf person to imitate it and to ascertain by identical procedure whether the stressing of any particular word has been correct or not. Not only is the speech of the deaf defective in the stressing of words but the entire sequence of speech is chopped off and devoid of its natural flow and continuity. For this deficiency, too, the vocal stimulator is an excellent remedy. The deaf pupil feels the rhythm of a sentence or a sequence of words said by his instructor on his own larynx and will imitate it. One must be aware of the fact that it usually takes many months of practicing before one succeeds in building up the new rhythmic patterns into the deaf pupil's spontaneous speech mechanism.

Since the vocal stimulator can be equipped with any number of larynx transducers, the apparatus may be used for the simultaneous speech instruction of any number of pupils. It is the number of larynx transducers which determines how many pupils can be stimulated at the same time. By means of a microphone and the transducers applied on his larynx, each pupil has the possibility of control of his very own speech rhythm, accent, and intensity.

I have had frequently the occasion to test the effectiveness of the vocal stimulator. The following case report may illustrate my experience with it.

REPORT OF A CASE

A 22-year-old woman was referred for speech improvement by an ear, nose, and throat specialist. She had been profoundly deaf since infancy. The circumstances which caused her deafness could not be determined. Her parents were rather wealthy and did for their child everything that could be done. She was quite attractive and pretty and wanted to get married, like any normal, healthy girl. However, there was a hitch to it: She did not want to marry a deaf man, although she had many suitors among her deaf friends.

In spite of all the speech instruction she had so far, her speech could not be understood by anybody, with the exception of her parents. Every young man she met stopped seeing her after the first date, because, although her skill in speech perception was excellent, her unintelligible speech made mutual communication impossible. She became somewhat frantic as to her curtailed marriage chances, and her parents took her eventually to the ENT specialist, who after a thorough examination recommended me for speech therapy.

When I first met and heard the patient, her speech could hardly compare with human speech. The sounds that came from this pretty girl's mouth resembled more the barking of a dog than a human voice. She distorted her face while speaking, and *soi disant* sentences were uttered with an enormous effort of the entire vocal apparatus. It was obvious that the patient was totally oblivious of the existence of rhythm, accentuation, and melody as being part of oral communication.

An analysis of the patient's speech revealed a guttural uncouth voice, marked rhinolalia aperta (nasal speech), and dyslalia # of a number of speech sounds; i. e., the "s" was completely omitted when combined with another consonant, such as "st," "sp," "str," "scr," etc. When isolated, "s" sounded like "t"; thus "scissors" and "sister" became something which sounded like "titto." The sounds "sh," "ch," and voiced "g" ("j") were substituted by sounds resembling "t" or "d." "K" and hard "g" (as in go) were replaced by noises which had no relation to the English language. No wonder that nobody could understand her and that every "hearing" young man was soon repelled by this ugly, incomprehensible jumble of sounds. Careful testing of her language patterns, however, gave evidence that sentence structure and grammar were perfect and that her vocabulary could favorably compare with that of any educated young woman.

During the first eight months of training, I did not yet have the vocal stimulator and had to confine myself to the customary methods of teaching speech to the profoundly deaf. From the very beginning I introduced breathing exercises,[35] with the goal of improving the patient's voice, and was fairly successful in eradicating most of the guttural tones of her vocal expression. Eventually I proceeded to attack the rhinolalia aperta.

Rhinolalia aperta frequently distorts the speech of the profoundly deaf. The speech of these deaf persons manifests the same nasal characteristics one encounters in people afflicted with the congenital deformity of cleft palate.[36] Although the oral cavity of the patient did not evidence any anatomic deformity, a physiologic deficiency could be noticed by the careful observer. While saying "ah" she did not raise her soft palate. The raising of the soft palate is a necessary movement to obtain the palatopharyngeal sphincter.[37] It is the palatopharyngeal sphincter which prevents the passage of air from the pharynx into the nasal cavities. Without the complete separation of the oropharynx from the nasopharynx normal speech is impossible. It seems, since rhinolalia aperta is so common in the speech of the deaf, that raising of the soft palate and the palatopharyngeal sphincter formation are learned functions which need the sense of hearing to be acquired for the speech mechanism. In this particular case, in order to correct the deficiency, I demonstrated to the patient the movements of my own palate, gave her a mirror and told her to try these upward movements with her own palate. Since she had difficulties in learning this movement, I lightly touched her soft palate with a tongue depressor and thus provoked some gagging. The gag reflex is accompanied by intensive contractions of the levator palati muscles. It is this muscular contraction which produces the upward movement of the soft palate, which is then drawn into contact with the ridge of Passavant. Eventually the patient became aware of this mechanism while saying the vowels. As a means of reinforcing the patient's kinetic control, I put her thumb and forefinger on my nose and let her feel the difference between nasalized and denasalized vowels. (No vibrations can be felt on the nares when English vowels are correctly enunciated.) By putting her fingers on her own nares she was able to determine whether the enunciation of her vowels was devoid of rhinolalia aperta. It was the denasalized enunciation of the vowel "i" (as in "if" and "meet") which proved to be most difficult for the patient.

Methods I have found to be effective for correction of dyslalias in cleft-palate speech [36] were applied to improve the enunciation of the sibilants. The small glass tube of an eyedropper placed at her

Dyslalia means the omission of a sound, the defective enunciation of a sound, or the substitution of a sound by another one.

lower incisors was a simple way of teaching her to blow the sounds "s" and "z." She succeeded in learning how to direct the air current through this narrow glass tube. The physiology of "s" and "z" requires sustained air pressure forced through a minute opening, an impossible feat in the presence of an open outlet between the pharyngeal wall and the soft palate.

A glass tube ½ in. (1.27 cm.) in diameter fixed her tongue in a central position in her oral cavity and enabled the patient to blow a concentrated "sh."

Step by step, the different dyslalias were corrected, and the patient applied them eventually in her spontaneous speech patterns. How to avoid distortions of her face while speaking had to be suggested very carefully to the patient in order not to hurt her feelings. The stressing of the right syllable in polysyllable words was beaten into the palm of her hand; i. e., the word "elementary" consists of two short, one long, and again two short beats. The entire process of speech education seemed very laborious, with no end in view. In spite of a number of improvements—a more pleasing voice, disappearance of the rhinolalia aperta, fairly correct enunciation of the consonants, and a better stressing of the individual words—a person unacquainted with the speech of the deaf could but have the impression of inadequacy of the patient's means of oral communication. The speech of the patient seemed to fall apart into incongruent pieces, with nothing to hold them together. In spite of all my efforts good intelligibility seemed still a far-away goal. My relationship with the patient became a very strained one. No young man dated her more than once, and she wanted to give up speech therapy altogether.

At this precarious moment, Mr. Nielsen let me have the vocal stimulator to use for my patients, and I persuaded the patient to remain a few weeks longer in therapy in order to try out the effectiveness of the new machine. After the first trial with it, she was convinced that the larynx transducers would help her to better organize her speech patterns. The vibrations of the transducers were amplified enough to stimulate her vocal cords, and the patient was able to emit tones in accord with these vibrations. For the first time in her life, she emitted tones devoid of any disturbing noises. In a relatively short period she learned to imitate five differently pitched tones which the transducers had amplified on her larynx.

By means of the vocal stimulator it became very easy to teach her the stressing of the different words and the rhythmic cadence of an entire sentence. "Practicing" became now the main problem, in order to accustom the patient to the newly learned speech rhythm. After three months of working with the vocal stimulator, her speech had at last lost all harshness and evidenced the natural "togetherness" which can be produced only by correct stressing, natural fluency, and rhythm. The patient's personal experience confirmed the improvements. She told me that people seem to understand her quite easily and that a dashing marine had dated her on several occasions. Two months later she confided in me that the marine seemed to have serious intentions. After five months of training with the vocal stimulator, the patient confessed that she was very busy preparing to get married. Since her fiancé had no trouble following her conversation, she did not see any need for further speech therapy. A few months later I received an invitation to the wedding.

I had worked, altogether, 13 months with this patient five times a week—8 months without the vocal stimulator and 5 with it. Considering the significant progress the patient made during the last five months, it can be reasonably assumed that if we had been able to work from the very beginning of the therapy with the vocal stimulator we could have achieved the same results in half the time.

SUMMARY

Aristotle with his pronouncement that those who are born deaf are senseless and incapable of reason inflicted 2000 years of punishment on the deaf. Not until the 16th century were the first attempts made to teach the deaf how to write. This gave them eventually the opportunity to express their thoughts by means of language.

Unfortunately, until very recently, the education of the deaf was split into two opposing camps advocating different methods of instruction. The French school emphasized instruction in sign or gesture language, whereas the German school concentrated on oral speech. In the United States the first schools for the deaf were organized at the beginning of the 19th century. Many educators of the deaf investigated very carefully whether instruction in oral speech or in sign language was to be preferred as the method of choice. The system which favors the oral method combined with signs is known as the "American combined system."

The Committee on the Deaf and Hard of Hearing of the White House Conference in 1930 worked out a terminology for the purpose of making an exact distinction between the profoundly deaf and the hard of hearing.

A systematic study concerning the residual hearing of the deaf was made in Vienna by V. Urbantschitsch, who advised hearing exercises for all deaf children. If after a period of practicing no hearing sensation can be elicited, hearing exercises have to be discontinued. Recent advances in electronics and in the physics of sounds made possible a considerable improvement in these techniques. According to studies made in schools for the deaf in London, Paris, and in the United States, approximately 40%-80% of these children may benefit from amplified speech.

Dr. Ruth Guilder, from the Clarke School for the Deaf, developed a very efficient clinical program adjusted to the remedial problems of the hard of hearing and the partially deaf child. This program includes individual instruction in speech correction, lipreading, and listening exercises with the benefit of a hearing aid. So far, in the schools for the deaf only partially deaf children with hearing losses in the speech range from 60 to 70 db. have been equipped with hearing aids. In ClarenceV. Hudgins' opinion, one should make use of the recently developed powerful hearing aids, by means of which even profoundly deaf children can be acoustically reached. The results of a series of experiments confirm Hudgins' assumption that even profoundly deaf children benefit considerably by the constant use of a powerful high-fidelity hearing aid.

Powrie Vaux Doctor, from the Gallaudet College in Washington, D. C., states, however, that of 21,545 pupils reported as being in schools for the deaf 10,110 cannot benefit from any kind of hearing aid. They have to be taught by vision and touch impressions.

The idea of communicating vocal vibrations instrumentally to the fingers or palms of deaf persons was executed by H. Gault. He trained his pupils to understand speech through the medium of touch impressions. Gault observed that by means of his method not only enunciation improved but also what he called accent, emphasis, rhythm, and tempo.

Without the knowledge of Gault's previous experiments, I was thinking along similar lines. One of my patients, Robert Nielsen, from the Bell Laboratory in Summit, N. J., following my suggestions, designed an apparatus which when brought into contact with a normal, healthy larynx picks up the vibrations of the vocal cords during phonation and transmits them amplified to the larynx of the deaf. These amplified vibrations proved to be a satisfactory stimulus to profoundly deaf persons and enabled them to emit tones similar to those they felt on their own larynges produced by means of the larynx transducers.

A case history illustrates the advantages gained through the application of the "vocal stimulator."

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Case Reports

ADENOCARCINOMA OF THE PAROTID DUCT

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and
PAUL K. HAMILTON Jr., M.D., Denver

MALIGNANT tumors of Stensen's duct are of rare occurrence. Goforth,[1] in 1927, reported a case of carcinoma which was successfully treated by radiation. Figi,[2] in 1944, described the case of an adenocarcinoma which was lost sight of. More recently, Smith and associates[3] published a detailed account of a parotid duct sarcoma which terminated fatally. To these may be added the following interesting cases.

REPORT OF CASES

CASE 1.—A middle-aged housewife presented herself on Nov. 5, 1934, with the history of having had a basal-cell carcinoma of the left forehead treated by cautery and radiation two years previously. Her present complaint was a painful lump in the left cheek of several months' duration.

On Nov. 13, 1934, with the area under local anesthesia, an adenocarcinoma approximately 1.5 cm. in diameter was removed by sharp dissection from the distal end of the left parotid duct. The loose end of the duct was sutured to the buccal mucosa, and patient was given full doses of radium.

In April, 1935, there were obvious metastases in the left submaxillary triangle which necessitated a suprahyoid neck dissection. After this, patient remained in good health for almost 20 years.

Subsequent history is interesting. A year and a half ago, patient was operated upon by Dr. James A. Stapleton for a melanosarcoma of the left forehead which terminated fatally. The occurrence of three types of malignancies in the same patient over a period of 20 years must be recorded as a pathological curiosity.

CASE 2.—A 58-year-old married woman was first examined on May 16, 1955. She complained of a painful swelling of the right side of her face which had been noticed for a few days only (Fig. 1). Other than that, her history was not contributory.

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Examination disclosed a hard but tender mass about 2 cm. in diameter in the substance of the cheek opposite the upper second molar. Adjacent to this mass a number of discrete nodules could be palpated beneath the buccal mucosa. Roentgenological and rhinological examinations were negative, and there was no palpable evidence of salivary calculi. With the exception of the distal end of the parotid duct, which was not clearly visualized, sialograms of the parotid gland and duct showed no abnormal shadows (Fig. 2). The flow of saliva from the ductal orifice was normal in amount and consistency; however, probing of the duct met with resistance and was followed by a trickle of blood. There was no palpable adenopathy in the cervical or jugular regions.

The discharge of blood following exploratory probing proved to be the most significant symptom in the differential diagnosis of this case. The parotid duct is a tough structure, the lining of which is not injured by careful instrumentation. Bleeding

Fig. 1 (Case 2).—Adenocarcinoma of right parotid duct simulating obstructive salivary calculus.

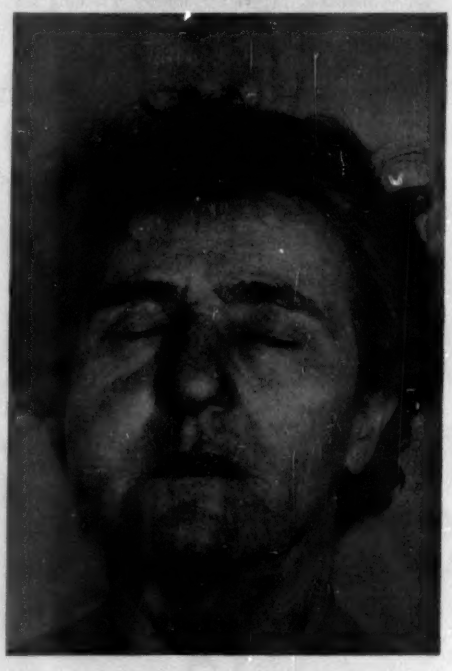
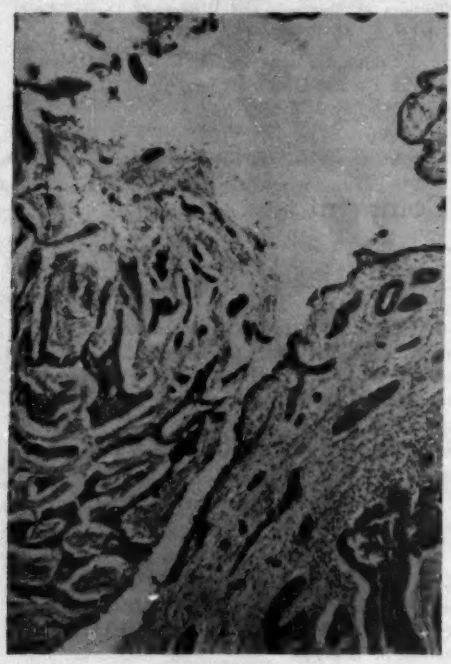


Fig. 2 (Case 2).—Sialogram.

Fig. 3 (Case 2).—Lumen of Stensen's duct is almost completely obliterated by this polypoid mass which is composed of anastomotic neoplastic cords that are apparently arising from the pseudostratified columnar epithelial lining of the duct. Hematoxylin and eosin; reduced about 1/9 from mag. × 100.



in the amount encountered here could hardly be attributed to trauma. In view of this finding alone, patient was sent to surgery with the tentative preoperative diagnosis of neoplasm of the parotid duct.

Operative Report.—On May 31, 1955, with the patient under thiopental (Pentothal) anesthesia, an intraoral incision was made, exposing a large tumor mass which completely surrounded the distal portion of the parotid duct. The duct was severed at the edge of the masseter muscle, and the mass, including a number of smaller nodules, was resected *in toto* by sharp dissection. No attempt was made to approximate the shortened end of the duct to the buccal mucosa.

Fig. 4 (Case 2).—Perineural lymphatics in the wall of the duct are distended with nests of neoplastic cells. Hematoxylin and eosin; reduced about 1/9 from mag. × 100.

Patient made an uneventful postoperative recovery. Surprisingly enough, nature has established a fistulous tract which accommodates the free flow of saliva from the parotid.

Gross Examination of the Specimen.—The specimen consisted of a segment of tissue composed of Stensen's duct and the periductal tissue. A probe was inserted in the ostium of the duct. However, it could be passed only approximately 1 cm. This segment of tissue was transversely sectioned from the ostium to the distal portion. One centimeter

from the ostium the duct was found to be almost completely obliterated by moderately soft tissue which extended from the wall. Within the wall of the duct in this area the tissue was gray and moderately firm, closely simulating the appearance of neoplastic tissue. A few small cysts were noted within this tumor-like tissue. Distal to this point the duct could not be identified.

Histopathologic Examination.—Sections of the duct through the area of obstruction revealed numerous irregular cords and nests of dark oval epithelial cells which had infiltrated the duct wall and formed a polypoid mass within the lumen of the duct partially occluding it (Fig. 3). The neoplastic cells were uniform, with oval hyperchromatic nuclei and a scant amount of cytoplasm. They formed acini in several areas and produced a mucinous material which was present in the lumen of the acini. Within the wall of the duct the perineural lymphatics were found to be invaded by the neoplastic cells (Fig. 4). There were focal areas of hyalinization within the stroma and also within several of the neoplastic cords. In several of the cords and nests of neoplastic cells two distinct cell types could be differentiated. In the basal layer the cells were small and moderately dark; in the outer layer the cells were larger and had large pale vesicular nuclei. The ductal epithelium of Stensen's duct was distinctly similar to the epithelial cells which made up the neoplastic nests and cords. In several areas a direct connection could be seen between the ductal epithelium and the neoplastic nodules.

Histopathologic Diagnosis: Adenocarcinoma (adenoid cystic carcinoma or cylindroma) of Stensen's duct.

SUMMARY AND CONCLUSIONS

The parotid duct is not a common site for malignancies. To the three reports from the literature, two cases of adenocarcinoma are added and described in some detail.

Case 1 survived surgery and radiation treatment for almost 20 years.

The predominant symptoms of Case 2, acute pain and swelling, suggested the presence of an obstructive stone rather than neoplasm. The serious character of the underlying pathology, however, was soon manifested by the free flow of blood coincident with exploratory probing of the duct.

The histogenesis of the presenting tumor is clearly demonstrated on microscopic section. Contiguity and similarity of the ductal epithelium to that of the tumor mass provide positive proof of the origin of this malignant lesion.

227 16th St.

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News and Comment

ANNOUNCEMENTS

Postgraduate Course in Otolaryngology.—The Department of Postgraduate Medicine of the University of Michigan Medical School announces the Otolaryngology Conference to be given at the University Hospital, Ann Arbor, Mich., on April 19, 20, and 21, 1956, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology at the University of Michigan Medical School.

Guest lecturers of national prominence, together with members of the staff of the Department of Otolaryngology, will participate in the program.

For further information, write to Dr. John M. Sheldon, Director, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Mich.

CORRECTION

In the Progress in Otolaryngology report in the November, 1955, issue of the Archives, "The Paranasal Sinuses," by Dr. Samuel Salinger, the name of the first author in Reference 61 (p. 558) was incorrectly given as Koch, B.; it should be Barr, B.

AN UNUSUAL CASE OF SPHENOID ABSCESS

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IT IS CERTAINLY true that sphenoiditis frequently brings to mind that "out of sight, out of mind." The literature is filled with reports of treatment and management of maxillary, frontal, and ethmoid sinus infections. The average rhinological examination can very well evaluate the status of these structures, but all too frequently the sphenoid sinus is somewhat overlooked in its proper evaluation.* And yet, there are no fewer than 13 vital structures that are intimately concerned with the average sphenoid sinus: the dura mater, the pituitary body, the optic nerve and chiasm, the cavernous sinus, the internal carotid artery, the abducens nerve, the oculomotor nerve, the trochlear nerve, the ophthalmic nerve, the maxillary nerve, the sphenopalatine ganglion, the sphenopalatine artery, and the pterygoid canal and its nerve. Certainly herniation of the mucosa through dehiscences in the bone has been known to occur. This may well extend the sphenoid sinus well beyond the normal limits of the sphenoid bone.† Unfortunately, a precursory examination of the nose may not always disclose the true condition of a nose, especially of the more remote sinuses.

The case to be reported is an example of faulty diagnosis and what it may lead to.

REPORT OF CASE

A 60-year-old white woman first developed right temporal, right eye, and right neck pain of a severe nature about February of 1953. She was taken to her local physician at that time. He said the nose was "negative," but x-rays of the sinuses showed a "dark place in the nose near the right eye." Shortly afterward, the right eye began to swell and to protrude. The patient was then taken to a competent neurosurgeon, and after careful examination it was felt that an aneurysm had developed behind the right eye. Arteriograms were not too definite, and after a week in the hospital the patient improved and was allowed to go home in March, 1953. The pain subsided and the eye receded. The patient was free of symptoms for over three months, but pain then recurred and the eye again began to swell. The patient was again taken to the neurosurgeon, who partially ligated the right internal carotid artery in the neck. Pain persisted in the right eye, and after some two weeks the artery was completely tied off. Pain was completely relieved for about three months. In February, 1954, the right eye again swelled up and the patient was again taken to the neurosurgeon, who found that the arterial tie had become loosened, and the vessel was again tied off. After this procedure the eye remained swollen and the right eye pain remained intense. Finally, in June of 1954, the patient had a craniotomy and the internal carotid was ligated inside the skull. There was no relief of the pain and the eye remained somewhat protruded. In August, 1954, the neurosurgeon again entered the skull and "clamped off a nerve." The pain persisted and the eye remained protruded. In December, 1954, the patient was taken through the entire State Hospital for a thorough examination. Little was found to account for the patient's symptoms, and she was dismissed as a hopeless neurotic and dope addict (she now required ⅓ grain [20 mg.] of Pantopon [a mixture of the opium alkaloids] every three hours for relief of pain).

In June, 1955, the patient was taken back to the original neurosurgeon and he did the third craniotomy with the plan of actually removing the portion of the cranial vessel that was thought to have contained the aneurysm. In doing this, a portion of the anterior clinoid process was accidentally fractured, opening up the roof of the sphenoid sinus on the right side. Thick yellow pus was seen filling this right sphenoid sinus, and the sinus and wound were immediately closed.

The patient was then brought under my attention and care. She was a poorly nourished white woman, chronically ill and in apparent pain. The eye was still somewhat protruded, and there was a paralysis of the right superior rectus muscle and

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From Baylor University College of Medicine.

* References 1, 2, 5, 6, and 7.

† References 3 and 4.

Fig. 1.—*A*, Water's view of sinuses. The metal clips of previous brain surgery are clearly seen, as is the site of the craniotomy.

B, Note the large thick-walled sphenoid sinus below and in front of the neurosurgical clips. Contents are cloudy.

C, Again note in the mentovertex position the large thick-walled right sphenoid is quite cloudy and the great amount of medial and lateral extension.

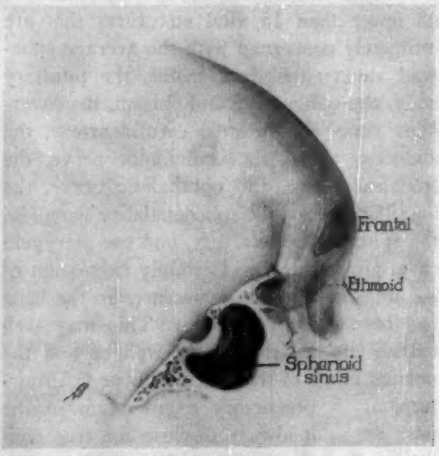

Fig. 2.—Artist's conception of the lateral x-ray view of the large right sphenoid sinus. Note the thick wall of the sphenoid and the absence of a natural ostium.

the levator muscle. Visual fields were normal, and the fundi were entirely normal bilaterally. The nasal examination, including careful nasopharyngoscopy, was entirely normal. No natural ostium could be found on the right side, although there was a bulge where the right sphenoid should be. The left sphenoid ostium was found and appeared normal. The ethmoids, although narrow, appeared normal, as did the frontal and maxillary sinuses. Sinus x-rays were made, including the 107 degree angle view of Granger, the mentovertex view of Hirtz, and a routine lateral view. This established without question that there was a large thick-walled right sphenoid sinus filled with a dense opaque material. In view of the history and the need for a good exposure, it was decided to make an external approach to the right sphenoid through the right ethmoid region. According to Proetz: "it is neces-

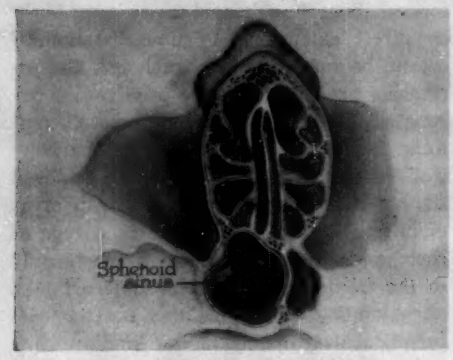

Fig. 3.—Schematic drawing made from the mentovertex position looking up from below. This is to illustrate again the amount of medial and lateral extension that this sphenoid had assumed. The reader's left is to the right of the patient.

sary to distinguish between an abscess and a pus filled sinus, a distinction not usually made in years past. The abscess is a collection of infectious material lying in an adventitious cavity, requiring evacuation and obliteration to return the tissues to normal. The sinus, on the other hand, is an infected hollow anatomical structure having functional parts temporarily deranged, but capable of restoration."[7] This structure with complete loss of ostium must be considered an abscess cavity serving no useful function and damaged beyond repair.

Accordingly, on July 24, 1955, with thiopental (Pentothal) sodium anesthesia, a curved external incision, about 1 in. long, was made ½ in. medial to the inner canthus of the right eye. The anterior ethmoids were entered, and next the posterior ethmoids were exenterated. There was practically no right frontal sinus. After the ethmoid labyrinth

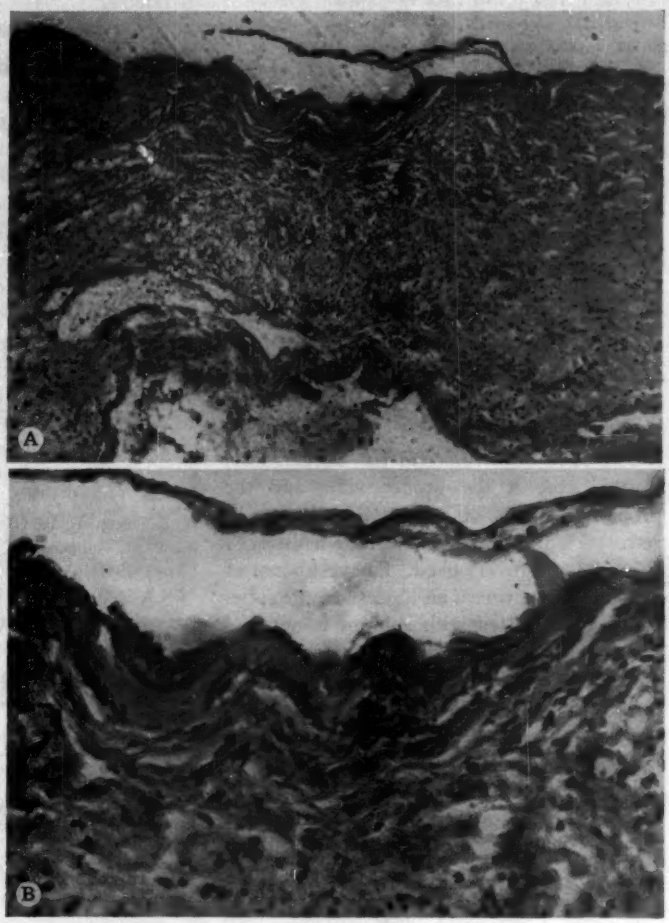

Fig. 4.—*A*, low power; *B*, high power. Tissue removed from the sphenoid cavity on the right. Note the thickness, with fibrous tissue infiltrated with inflammatory cells. One area of actual abscess formation can be noted. It was felt that in this case the lining membrane should be removed to completely clean out this sphenoid sinus.

was cleaned out, a firm bony resistance was met where the right sphenoid sinus should have been. It was necessary to use a small gouge and mallet to enter the right sphenoid sinus. The bone was fully ¼ in. thick and gritty to the bite. A large sinus cavity filled with pus, debris, and thick cheesy material was discovered. The entire anterior wall of the structure was removed as high as the level of the dura and as low as possible. The sinus extended well over to the left side of the septum of the nose and far laterally. When eventually cleaned out, it contained in excess of 15 cc. of liquid. The gangrenous necrotic lining was carefully removed, revealing rough bone. The roof of the sphenoid was not touched in as much as this was the site of the original accidental entrance from the cranium. Bleeding was slight and easily controlled. Packing was inserted, and the patient tolerated this procedure quite well.

At the present time the patient still has some pain, but during the past six weeks it has gradually become less, and she is being treated for her drug addiction. Her personality is also improving.

COMMENT

This unfortunate patient was the victim of a misdiagnosis for two and one-half years. Certainly this was a case of "out of sight, out of mind." The neurosurgeon depended on a previous sinus examination (in fact, by two different teams of examiners) to give the proper evaluation of the presence of sinus disease. The usual methods of examination—(1) direct inspection, (2) nasal smear, (3) nasopharyngoscope, and (4) x-ray of the sinuses—had been done. Diagnostic lavage in this case was impossible. However, x-ray should have been sufficient for the diagnosis in this case if the proper view and the proper interpretation had been employed. In retrospect, it is hard to see how a diseased sphenoid was overlooked. I have known of one other case where an abscess of the sphenoid was confused with a brain tumor and the patient died after an exploratory craniotomy. The most helpful x-ray views in this case were (1) the routine lateral view of the skull and (2) the Hirtz, or mentovertex, position.[6]

In reviewing the history and findings of this case, it is now felt that a chronic right sphenoiditis developed, probably years ago, and as the condition slowly progressed a proliferative osteomyelitis (and osteitis) formed about the sphenoid cavity, most likely by direct extension. In time the natural ostium became completely occluded, and increasing pressure and bone proliferation accounted for the prolonged and severe symptoms. The microscopic report of the bony wall of the sphenoid which was removed showed much new-formed bone with areas of infection scattered throughout, a type of chronic bone infection seen not uncommonly in the frontal sinuses when subjected to a long-standing infection. It is not too surprising that symptoms of this kind are slow to recede. It was felt that actual obliteration or ablation of this sphenoid abscess should be accomplished in so far as was possible. Any less radical procedure would be doomed to failure.

SUMMARY

A case of sphenoid abscess is presented where direct examination of the nose was largely normal. No evidence of pus or inflammation was seen preoperatively. All of the means at our command should be used in cases of this type to rule out sinus disease. In this case proper x-ray pictures and interpretation (Fig. 1) were most important. Many unnecessary operations and dope addiction could have been avoided. This instructive case should make us all hesitate to state blandly that "the sinuses are negative."

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THE MANAGEMENT OF LARYNGEAL KERATOSIS BY LARYNGOFISSURE

ARTHUR J. CRACOVANER, M.D., New York
and
ALAN S. RUBENSTEIN, M.D., Wilkes-Barre, Pa.

KERATOSIS of the larynx is seen rather frequently in a large laryngeal clinic and is often a perplexing disease from the standpoint of treatment. During the past several years, we have encountered cases at the New York Eye and Ear Infirmary that eventually came to operative removal by laryngofissure, and the reasoning involved in such a conclusion is the purpose of this communication.

Keratosis or hyperkeratosis of the larynx is a localized hyperplasia of the epithelium. Grossly, it appears as a white or grayish-white elevated area that may be smooth but usually is irregular and wrinkled. Its elevation makes it stand out from the surrounding mucsoa, which very frequently is in a state of chronic inflammation.

Microscopically, keratosis is recognized by an increase in the size of the squamous epithelial layer, especially of the cornified portion. The surrounding mucosa and submucosa sometimes show evidence of a chronic inflammatory process. However, if cells with atypical nuclei of malignant tumors are recognized in the mucosal layer, the specimen is considered carcinoma in situ, and, if the basement layer is broken by infiltration of these atypical cells, the diagnosis is infiltrating carcinoma of the larynx.

In the treatment of small keratotic lesions of the larynx, we usually remove the lesion with forceps by direct laryngoscopy and hope to eradicate the entire disease process. Sometimes we strip the entire vocal cord of its mucous membrane, for the lesion may involve the whole vocal cord or may be multiple. It is important that the patient refrain from smoking and drinking alcoholic beverages. However, all too frequently the lesion has a tendency to recur, and the patient must be kept under constant observation for this possibility. It may be necessary to remove recurrent growths several times. It then seems wise to ensure complete removal by resecting the cord through a laryngofissure operation.

Occasionally, there are lesions that are rather large, having a broad base and involving not only the vocal cord but the ventricular area and/or the subglottic area. They are so extensive that removal with forceps is impossible and laryngofissure is necessary.

Some of these large tumors clinically appear to be carcinomas but microscopically are reported as hyperkeratotic lesions. We cannot always be certain that biopsy specimens have included areas that are malignant, and so these large broad-based lesions must be treated as malignant lesions and removed through the thyrotomy approach.

Keratotic lesions, small or large, are sometimes reported as "suspicious" or as containing "atypical cells." This may occur on the first removal by direct laryngoscopy or after several removals. These are to be considered malignant and must be completely resected by laryngofissure.

Then, there are the hyperkeratotic lesions that definitely reveal microscopically the presence of malignant cells. It goes without saying that these are treated as is any other malignancy of the larynx. According to its extent, a laryngofissure or, in some instances, a laryngectomy is required.

Received for publication Sept. 28, 1955.

From the Department of Bronchology and Laryngeal Surgery, New York Eye and Ear Infirmary.

REPORT OF CASES

CASE 1.—A man, aged 63, was first seen on May 11, 1953, with the chief complaint of hoarseness of

three months' duration. Mirror examination of the larynx revealed a papillomatous lesion on the midportion of the right vocal cord. Both cords were normally mobile. A chest x-ray taken on May 13 showed a sharply circumscribed infiltrative lesion in the infraclavicular portion of the right lung, suggestive of an old tuberculous process. On May 14 biopsy was performed by direct laryngoscopy, and the pathologic diagnosis of one of the two pieces of tissue submitted for study was "squamous-cell carcinoma, Grade 2, of vocal cord, arising in hyperkeratosis." (Fig. 1.) The other specimen showed only hyperkeratosis. On May 21, laryngofissure was performed and a lesion on the anterior one-half of the right vocal cord was removed. The pathologic report was keratosis of the larynx, no carcinoma being found in the laryngofissure specimen. Aside from a small amount of subcutaneous emphysema, the patient had a normal postoperative course, and he was discharged on June 3, with his tracheotomy wound almost completely healed. The patient has been observed repeatedly, and there has been no recurrence of any lesion in the larynx.

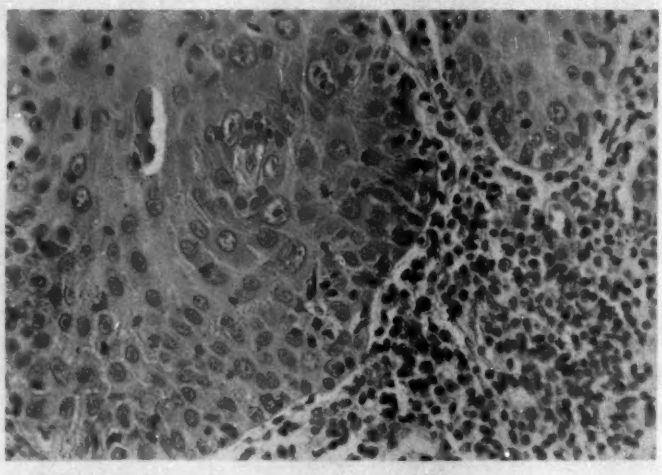


Fig. 1 (Case 1). — Photomicrograph showing the intraepithelial cancer cells on the left and the definite carcinomatous infiltration on the right. The stroma is densely infiltrated by lymphocytes.

CASE 2.*—A man, aged 55, was admitted to the New York Eye and Ear Infirmary on Jan. 27, 1954. Prior to this time the patient had had at least five endolaryngeal removals of polypi and keratotic lesions; two years previously, he had had a sudden attack of suffocation while driving his car and had undergone an emergency tracheotomy at another hospital. The patient had continued to the present to be hoarse, and he noticed increasing laryngeal dyspnea. Mirror examination revealed a large grayish-white cauliflower lesion in the glottic lumen, apparently arising from the anterior portion of the left cord subglottically. His blood pressure was 170/90, and the remainder of the physical examination and laboratory findings were normal. On Jan. 28, laryngofissure was performed and a large, sharply circumscribed, elevated, whitish lesion was removed from the inferior surface of the left vocal cord and the subglottic area. The pathologic diagnosis was hyperkeratosis of the larynx, with no anaplastic changes noted (Fig. 2). The patient had a good deal of subcutaneous emphysema over his chest and face for a few days following his operation, but otherwise his course was uncomplicated, and he was discharged on Feb. 4.

CASE 3.—A man, aged 46, admitted to the hospital May 18, 1954, had a long history of hoarseness. Direct laryngoscopic biopsies had been done on Feb. 3 and 13, 1954, and the specimens revealed keratosis of the right vocal cord in each instance. The lesion had been observed to recur and had extended to involve the entire right cord. Mirror examination on this admission revealed a white irregular keratotic lesion involving the right vocal cord from the anterior commissure to the vocal process. The left vocal cord was clear, and both cords were mobile. On May 19, laryngofissure was performed and a large cauliflower lesion was removed from the right vocal cord and the anterior portion of the left cord. Several hours following the operation persistent hemorrhage from the wound necessitated return of the patient to the operating room for ligation of bleeding vessels in the depths of the tracheotomy wound. The patient had a temperature elevation to 102 F rectally on May 19 and 20, but his postoperative course was uncomplicated thereafter. The patient was discharged on May 26 and had had no recurrence of his disease one year

* Dr. Howard Bancks has given us permission to include this case.

Fig. 2 (Case 2).—The oblique sectioning of the epithelium emphasizes the keratosis. Note the absence of significant cell atypism in the squamous epithelium but the presence of lymphocytic response in the underlying tissues.

later. Pathologic diagnosis of the laryngofissure specimen was hyperkeratosis, with a large amount of inflammatory reaction (Fig. 3).

CASE 4.—A man, aged 69, was admitted to the hospital on May 19, 1954, with a history of hoarseness for three months. Mirror examination in the clinic had revealed a whitish lesion on the right vocal cord, with fair movement of this cord. Direct laryngoscopy on May 20 revealed a large keratotic-appearing lesion of the entire right vocal cord and subglottic area. Several biopsy specimens were submitted and were reported as showing hyperkeratosis with suspicious areas of cellular activity and considerable inflammation (Fig. 4). The clinical impression from the appearance on direct laryngoscopy was carcinoma engrafted on keratosis. On May 26, laryngofissure was performed and a large fungating lesion was removed from the anterior one-third of the right vocal cord, along with a portion of the ventricular band. The postoperative course was smooth, except for infection and granulation production of the tracheotomy wound. This responded readily when dressings of Peruvian balsam and castor oil were used and tetracycline (Achromycin) hydrochloride was given orally. The patient was discharged on June 7 and has remained well to the present. The pathologic diagnosis of the laryngofissure specimen was carcinoma in situ in a hyperkeratotic lesion. It was believed that the carcinoma was removed entirely, and the patient had had no recurrence for 11 months following thyrotomy. However, he subsequently died of a carcinoma of the right upper lobe bronchus with metastases.

CASE 5.—A man, aged 52, was first seen in the clinic on Aug. 17, 1939, with the chief complaint of hoarseness of four years' duration. Mirror exami-

Fig. 3 (Case 3).—Regular and orderly but excessively cornified squamous epithelium from the vocal cord. The underlying stroma does not appear infiltrated.

nation at that time revealed a white irregular mass involving the right vocal cord, while both cords moved freely. On Aug. 23, biopsy was done and the pathologic report was papilloma. On Sept. 19, another biopsy was performed and a large white papillomatous mass was removed from the anterior commissure of the right vocal cord. Pathologic diagnosis was again papilloma. The growth continued to recur, and from its appearance mycotic disease was considered. Many tissue cultures on appropriate media were carried out, but no fungi were identified. Another biopsy was performed on Nov. 21, and papilloma with hyperplasia of the epithelium, with some downgrowth, was seen on microscopic study. From the latter part of 1939 until 1941 the larynx was treated by various topical methods, particularly with the application of 10% patient has been observed for five years following thyrotomy, and there is no recurrence of the lesion.

CASE 6.—A man, aged 60, complained of hoarseness for "several months." He was a heavy smoker and drinker. On examination, a glistening whitish lesion was found covering the anterior two-thirds of the left vocal cord and reaching the anterior commissure. The cord was mobile. The right vocal cord seemed uninvolved. On April 14, 1955, direct laryngoscopy and biopsy were performed. The microscopic report was "hyperkeratosis with some cell atypism; malignant transformation not evident." Because of the extent of the lesion and its character, laryngofissure was done on April 19 and the left vocal cord was removed. However, the anterior end of the right vocal cord appeared a little thickened and a portion of this was also removed. The patient

Fig. 4 (Case 4). — Changes similar to those in Case 1. Note the regularity of the epithelial cells, the keratinization, and, in this area, the moderate to slight lymphocytic reaction.

neoarsphenamine on glycerin. On Oct. 1, 1940, Jan. 7, 1941, and Feb. 25, 1941, biopsies were again performed, but the pathologic diagnosis was unchanged, and no fungi were cultured. From 1941 until 1950, the patient apparently remained away from the clinic and evidently was fairly well during this period of nine years. In March, 1950, he returned with increased laryngeal complaints, and direct laryngoscopy on March 9, 1950, revealed a large ulcerated lesion extending from the anterior commissure of the right vocal cord to the vocal process. A biopsy specimen was taken, and the pathologic impression was hyperkeratosis, strongly suggestive of malignancy. On March 23 yet another biopsy specimen was taken, which showed only keratosis, no carcinoma. On April 20 laryngofissure was performed, and the postoperative course was uneventful. The pathologic diagnosis was hyperkeratosis of the vocal cord, with no carcinoma. The made an uneventful recovery. The pathologic report stated: "Left vocal cord and adjacent tissue: leukoplakia with associated carcinoma in situ; no infiltration in evidence. Right vocal cord: leukoplakia of slight to moderate extent."

COMMENT

From these cases, and from others not reported, we would conclude that laryngofissure for keratosis should be performed earlier than has been our custom heretofore. We have performed numerous biopsies on our patients with equivocal reports from the pathologists. We have apparently removed carcinoma in situ by biopsy (Cases 4 and 1). The patients have been subjected to repeated hospitalization, continued worry and uncertainty, and perhaps to needless jeopardy

through our procrastination. Laryngofissure has almost always demonstrated an elevated, hard lesion with a very broad, firm attachment. All the endolaryngeal cord-strippings in the world would not have removed these lesions in their entirety. Yet the sharply defined nature of the keratosis made surgical excision of the lesion by thyrotomy simple, complete, and most satisfying. The patient's postoperative voice is not usually much different from the preoperative, since these patients with extensive keratosis are very hoarse. All told, after medical and endolaryngeal procedures have failed, the management of keratosis by laryngofissure has been very successful at the New York Eye and Ear Infirmary.

SUMMARY

Keratosis of the larynx is often impossible to cure by medical means or by endolaryngeal methods. In a very definite proportion of cases diagnosed as keratosis of the larynx the lesion will become malignant or is actually malignant from the start. Laryngofissure is an ideal method of management when more conservative measures fail, and it should be used more frequently and probably earlier than has been our custom formerly.

114 E. 72d St. (Dr. Cracovaner).

Dr. George K. Higgins helped in the pathological aspect of this paper.

News and Comment

ANNOUNCEMENTS

Sixth International Congress of Otolaryngology.—The Sixth International Congress of Otolaryngology will be held in Washington, D. C., from Sunday, May 5, through Friday, May 10, 1957. Selected subjects for the Plenary Sessions will be (1) Chronic Suppuration of the Temporal Bone, (2) Collagen Disorders of the Respiratory Tract, and (3) Papilloma of the Larynx. Outstanding internationally recognized authorities will open the discussions on each of these subjects. Communications of two types are invited: contributions to the discussion of the selected subjects (speakers limited to 5 minutes) and original papers (speakers limited to 15 minutes). All communications should be in one of the official languages: English, French, German, Spanish. Motion pictures will be shown continuously except during Plenary Sessions. There will be both scientific and technical exhibits. Those wishing to submit contributions to the program should communicate with the General Secretary.

Announcement of the Congress has been sent to all otolaryngologists whose names and addresses could be obtained. Additional details concerning registration, housing, entertainment, etc., will be sent to those who have indicated to the General Secretary that they wish further information.

The subscription for Members (physicians) is $25.00 U. S. A. This includes the privilege of attendance at all official Congress meetings except the banquet, for which an additional charge will be made. Other persons accompanying Members may be registered as Associates at a fee of $10.00 U. S. A.

An interesting program of social functions, visits to points of interest in and around Washington, and post-Congress tours is being arranged. The American Express Company is the official travel agent for the Congress. Their offices throughout the world are available for travel arrangements to the Congress and for post-Congress tours.

All communications should be addressed to the General Secretary, Paul H. Holinger, M.D., 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

A CHLORPROMAZINE (THORAZINE)-INDUCED CEPHALALGIA AND ITS MANAGEMENT

SAM E. ROBERTS, M.D., Kansas City, Mo.

INTRODUCTION

MY REASON for reporting this case is that chlorpromazine hydrochloride (Thorazine) is a relatively new preparation, is widely used, and, therefore, side-effects should be carefully noted. In the main this drug is well borne. In this case, however, it produced an excruciating head pain which could be controlled only by another preparation, which had an opposite physiologic effect.

REPORT OF A CASE

A 33-year-old white woman, a typist, was admitted to the Research Clinic, April 18, 1955, with chief complaints of pain in the back, hips, legs, and chest of 12 years' duration, following a spinal accident.

She had also noted marked paresthesia of both feet and toes, worse on the right side. She had not been subject to headache, but her mother had migraine, which was relieved by the inception of the menopause.

The findings of the clinic were essentially negative except for a scoliosis of the lumbar spine and a spina bifida occulta, the surgical correction of which, on May 24, was without incident and noncontributory to this report.

On June 7th patient was allowed to sit up with a back support. She complained of a mild headache, controlled by acetylsalicylic acid (aspirin) and codeine.

On June 17th the internist noted that the paresthesia was unimproved. He ordered chlorpromazine, 25 mg. t. i. d. As stated above, the patient did have some headache before the chlorpromazine was started, but the intensity increased daily with medication, until it became unbearable. The usual antipyretic analgesics with codeine gave no relief, and meperidine (Demerol), 75 mg., several times daily only stupefied the patient but did not relieve the pain.

I saw her June 22nd. It seemed reasonable that the chlorpromazine, a powerful vasodilator, was probably a strong contributing etiologic factor in her excruciating pain. The fact that the pain was not relieved by powerful opiates seemed to confirm the diagnosis of a vascular (vasodilating-type) headache, notoriously resistant to any therapeutic agent except a vasoconstrictor.

I ordered chlorpromazine discontinued and ergotamine with caffeine (Cafergote) every hour the afternoon of June 22nd. The relief was minimal, if at all. The morning of June 23rd the neurosurgeon ordered 200 cc. of 0.1% procaine, intravenously; and I ordered ergotamine with caffeine, to a total of eight in 24 hours.

The afternoon of June 23rd the patient was raving with the pain and imploring the nurse to give another stupefying opiate. To eliminate any psychogenic influence, I ordered the injection of a placebo. The pain, however, continued to become more intense. At 5:30 p.m. I ordered dihydroergotamine (D. H. E. 45), intravenously; and at 6 p.m. she was relieved and had a comfortable night.

At this point the following postulations seemed reasonable: 1. It was a vascular (vasodilating-type) headache. 2. The pain was probably produced by chlorpromazine, a powerful vasodilating preparation, and relieved only by a powerful vasoconstricting agent, dihydroergotamine.

The thought remained, however, that the proof was not entirely convincing, since the neuro-osseous spinal mechanism had undergone surgical trauma, which was preceded by a myleogram, both of which might be contributing factors. For academic purposes, I asked permission of both the neurosurgeon and the internist to be allowed to attempt to reproduce the same pain syndrome with the same pharmacologic agent, and in the same manner.

This privilege was granted to me, and on June 27th the chlorpromazine was again administered. That afternoon the patient complained of some pain, but it was fairly well controlled by aspirin and codeine.

Accepted for publication Aug. 4, 1955.

The following afternoon, however, after the second dose of chlorpromazine the pain again became unbearable.

The chlorpromazine was promptly discontinued, and 1 cc. of dihydroergotamine was given intravenously at 5:30. At 6 p.m. the pain was entirely gone; no more pain-relieving agents were required.

COMMENT

The above case report seems to show quite conclusively that chlorpromazine (Thorazine), a potent vasodilating agent, is also capable of producing the severest type of cephalalgia, closely resembling in intensity that of histaminic cephalalgia (Horton's syndrome), but without the joyous remissions of pain which characterize Horton's syndrome.

I believe any psychogenic factor was eliminated with the placebo. The fact the identical type of pain was reproduced by the readministration of the same vasodilating agent and controlled by the identical vasoconstrictor seems to leave little doubt that the original postulations are now factual.

1110 Professional Building.

Cooperation and helpful suggestions were given by Dr. Revis Lewis, neurosurgeon, and Dr. Frank Leitz, internist.

News and Comment

ANNOUNCEMENTS

Fifth Annual Scientific Session.—The Brooklyn Eye and Ear Hospital Alumni Association will present its Fifth Annual Scientific Session. It will be held at the Brooklyn Eye and Ear Hospital on Saturday, April 7th, 1956.

The program that has been arranged is as follows:

OPHTHALMOLOGY

Dr. Ralph I. Lloyd	Glaucoma in 1900
Dr. Willis S. Knighton	Medical Treatment of Glaucoma
Dr. Harold G. Scheie	Surgical Management of Glaucoma
Dr. Daniel Kravitz	Gonioscopic Significance in Glaucoma Surgery

There will be a motion picture symposium on the following subjects:

1. The Surgical Treatment of Glaucoma
Goniotomy and Goniopuncture in the Treatment of Congenital Glaucoma..Dr. Harold G. Scheie
2. Cataract in Glaucoma (loaned through the courtesy of Dr. Wendell L. Hughes)

OTOLARYNGOLOGY

Dr. Philip Meltzer	Surgical Treatment of Deafness
Dr. N. Lagoumis	A Series of Case Reports of Peripheral Facial Nerve Paralysis
Dr. Salmon Harvey	Physical Aspects of Hearing and Equilibrium as Applied to the Human Ear
Dr. L. A. Peer	Plastic Surgery of Malformations of the External Ear
Dr. Irwin Fine	Differential Diagnosis and Treatment of External Ear Disease

Basic Science Course in Otolaryngology.—The Department of Otolaryngology of Washington University, St. Louis, is offering a Basic Science Course in Otolaryngology, to start on Monday, Sept. 17, 1956.

Complete information about the course may be obtained by writing to The Head of the Department of Otolaryngology, 640 S. Kingshighway, St. Louis 10.

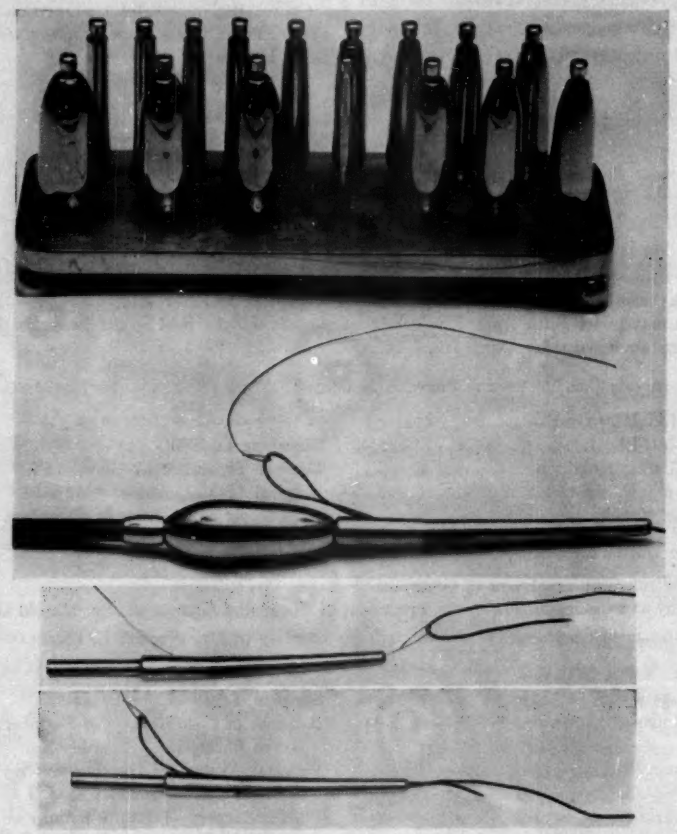
Clinical Notes, New Instruments and Techniques

ESOPHAGEAL DILATOR

A Simple Method for Threading

HARVEY H. SIROTA, M.D.
AND
VERA P. SHEPPARD, R.N.
East Hartford, Conn.

When the Jackson-Plummer dilating set for esophageal stricture is used (Fig. 1), the patient must first swallow approximately 4 yd. of 00 silk. At the time of the dilation, the exposed end of the swallowed silk is introduced or threaded through the perforated flexible-metal spiral tip which is used as a guide for the whale-bone staff threaded with sippy, conically shaped olive dilators. The threading of the silk through the spiral tip can be a difficult step, time-consuming,



laborious, and annoying. Although other methods will eventually succeed, we have found the following method simple and most convenient:

Take an 8 in. length of No. 4 wire (nasal snare wire) and make a small eye at one end by looping it over. Pass the other end of this wire through the perforation in the spiral tip and, with the silk threaded through the loop (Fig. 2), pull the wire through. The silk thread comes through with the wire, and the instrument is ready for operation (Fig. 3). The wire can also act as a stylet, cleaning and keeping clear the perforation of the spiral tip.

1128 Main St.

Accepted for publication Sept. 30, 1955.

OTOMETER (A NEW PORTABLE HEARING TESTING INSTRUMENT)

HOWARD P. HOUSE, M.D., Los Angeles

There has long been need for a small compact portable air conduction testing device that can be used in the home, hospital, and operating room (stapes mobilization). Such an instrument may also be of value in industry and for clinicians working in fields other than otolaryngology.

With this in mind certain specifications which I suggested were incorporated in an air conduction testing device known as on "Otometer." The specifications are as follows:

Frequencies: Sine wave tones of 500, 1000, 2000, 4000, and 6000 cps are available with an accuracy within ± 5%.

Tone interrupter: A momentary "on" switch with a bear knob is incorporated as a fast-acting interrupter switch, and the circuit is designed in a manner that no transient or extraneous frequencies are emitted when the tone interrupter switch is operated.

Otometer for air conduction testing.

Panel meter: A meter is incorporated in the Otometer with push buttons and rheostats for the A and B batteries for accurate control of the battery voltage.

Inasmuch as the instrument normally operates at two-thirds the battery voltages, the usable life of the battery is increased proportionately. By means of the battery voltage control adjustments and the meter, the proper voltages will always be supplied to the instrument. The estimated life of the B battery is at least one year, and that of the A battery, six to nine months.

The Otometer is furnished complete with a headband and a properly electronically calibrated dynamic receiver and cushion.

The weight of the unit is less than 3 lb. and measures in the carrying case 9 by 6 by 4¼ in. Unit dimensions are approximately 6 by 5 by 3 in.

Inasmuch as the instrument sells complete for a fraction of the cost of a complete audiometer, I believe that for specific purposes the Otometer will be in demand in many areas. The Otometer is manufactured by A. M. Brooks Company, 1222 W. Washington Blvd., Los Angeles 7.

1136 W. Sixth St. (17).

Received for publication Sept 6, 1955.

A BILATERAL SELF-RETAINING NASAL SPECULUM

MAX UNGER, M.D., New York

I wish to submit to the profession a bilateral self-retaining nasal speculum for use in septum resection operations. I have tested it by many years' usage and found it to be an extremely useful instrument.

The speculum is made of spring wire. It consists of two individual specula, a right and a left, that are connected above to form one unit, as illustrated in Figure 1. *H* in Figure 1 is a hook attached to the upper extremity of the instrument, and the upper end of the hook is hooked over

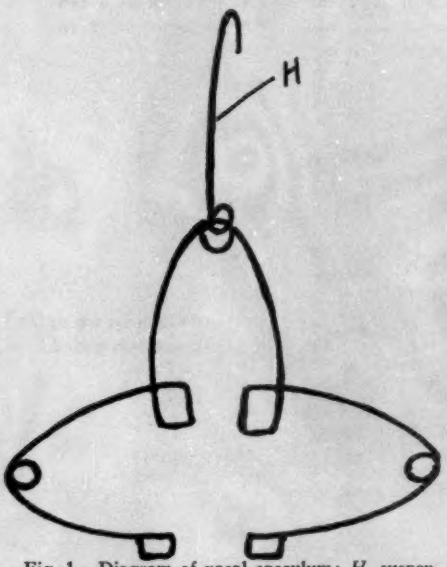


Fig. 1.—Diagram of nasal speculum; *H*, suspension hook.

Fig. 2.—Bilateral self-retaining nasal speculum in position.

a bandage that is tied across the patient's forehead. Figure 2 shows the speculum in use on a patient and gives a good idea of the manner in which it is inserted into the nostrils and suspended from the band across the patient's forehead. Figure 2 also shows the exposure that it gives. When properly made and inserted, the instrument can stay in place in spite of considerable intranasal instrumentation.

36 W. 40th St. (18).

Received for publication Sept. 26, 1955.

BRONCHOSCOPIC BIOPSY SPOON

G. ZAKHARY, M.B., Ch.B., D.L.O.
Cairo, Egypt

What made me think to design a new surgical instrument for taking biopsy specimens from the intrabronchial growths is the high percentage of negative results in specimens taken by the ordinary biopsy forceps, especially in cases where there is a growth in either upper lobe bronchus. This instrument has its value mainly in taking biopsy specimens from both upper lobe bronchi, and I do not advise using it in taking specimens from the esophagus, as the probability of injuring the mucous membrane of the esophagus in using it is more than that with the ordinary biopsy forceps, owing to the difference in the anatomical features of the bronchus and esophagus. Apart from its use in taking specimens, its handle can fit cauterizing ends of high-frequency currents (diathermy). This has its value in all lesions that need intrabronchial or intraesophageal electrocoagulation, for example, excessive intrabronchial bleeding after a biopsy specimen is taken which does not respond to ordinary measures.

Also, in cases of bronchial stenosis due to healed tuberculous ulcerative bronchial lesions I do lineal cauterizations at right angles to the fibrous tissue bands forming the stenosis. This results in the formation of regular bands of fibrous tissue which pull on the original bands, leading to a moderate degree of widening the stenosed lumen of the bronchus.

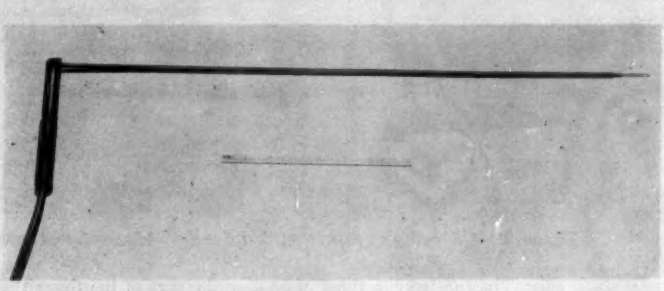


Figure 1

The last procedure should be done by expert hands, and, to obtain more successful permanent results, pneumatic bronchial dilatation should be carried out not before seven days from the date of cauterization.

For this reason I have designed different shapes of cauterizing ends that fit the direction and shape of the stenosis (Fig. 3). I am not going to discuss all the indications of intrabronchial electrocoagulation in this paper.

As regards esophageal lesions, one of the values of this instrument is in the cauterization of varicose veins in the distal third of the esophagus, which sometimes result in severe hemorrhage.

The lumen of either an adult bronchoscope or esophagoscope can easily accommodate both the instrument and the sucker tube, and so all the manipulations can be done under vision, a fact which makes this procedure very safe and accurate.

DESCRIPTION

The instrument consists of the following parts: There is a handle which is almost a right-angled one, with a vertical limb which is about 10 cm. long, sufficient for a gentle hand grip. At the bottom of that limb there is an electrically isolated yellow-copper socket in which the plug of the diathermy wire can be easily fitted if the instrument is going to be used for the purpose of cauterization; when the instrument is being used to take a biopsy specimen no wire should be plugged in. The upper end of that limb, where it joins the horizontal limb, is shaved on both sides so that its thickness does not obstruct the vision of the surgeon.

The horizontal limb is about 50 cm. in length, that is to say, a few centimeters longer than the adult-size bronchoscope or esophagoscope; it is about 3 mm. in diameter, and it is semiflexible; this has its value in diminishing any trauma to the mucous membrane of the bronchi, as it yields to a reasonable degree through the different surgical manipulations. At the free end there is a hole about 1 cm. deep, in which the cutting or cauterizing ends can be fitted safely, as they are screwed in for a distance not less than 8 mm. Owing to this mechanism, there is no danger of any of these ends slipping inside the lumen of the bronchus or esophagus.

Biopsy Cutting Ends.—Different ends have been designed in addition to the one illustrated in Figure 2.

Other Ends.—(1) Cutting fork end; (2) biangular cutting end with a hook, and (3) straight, curved, rounded, or oval curettes with sharp or blunt edges.

Mechanism and Construction of the Cutting Ends.—The end has a length of 2 to 3 cm. that is formed of three parts: (1) screw end; (2) central part, which is about 2 cm., made of spiral malleable metal with a spring action (Fig. 2); (3) cutting end, made of stainless steel. The one illustrated is the one mostly used by me; it has a champagne-cup shape, with a sharp

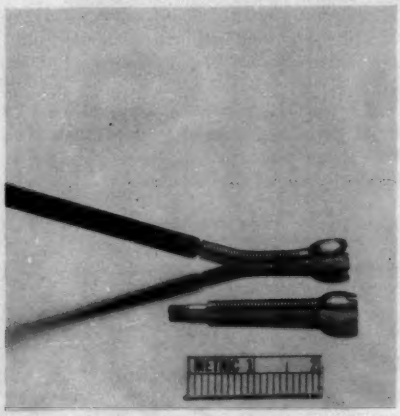


Figure 2

Figure 3

cutting castle-shape edge. The cup has the value of holding the part of the tissue that was cut by the edge.

COMMENT

The whole handle and the cutting end work in a leverage action with a slight rotatory movement.

Dr. H. J. Moersch, head of the bronchoesophageal section at the Mayo Clinic, became interested in the instrument and had a copy made of it. He is giving it a clinical trial.

Mayo Clinic has given assistance with my lantern slides.

A report of this instrument was presented before the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 12, 1955.

80 Gamhorie St.

ADDENDUM

A new modification of the bronchoscopy biopsy spoon consists of connecting the handle of the instrument to an electrical vacuum pump while the specimen is being taken. This helps hold the piece of tissue cut by the cup end. The suction is controlled through a hole at the angle formed by the junction of the handle with the horizontal limb, by the thumb of the surgeon.

Abstracts from Current Literature

Ear

OTOSCLEROSIS. TERENCE CAWTHORNE, J. Laryng. & Otol. **69**:437 (July) 1955.

The Dalby Memorial Lecture presented by Terence Cawthorne reviews in an excellent manner the subject of otosclerosis. Cawthorne states that the cause of otosclerosis is unknown and that we do not know why it attacks only the bony capsule of the labyrinth and is found more frequently in women. The subject is covered in an orderly and interesting fashion. General discussions of the subjects of hearing tests, classification of otosclerosis, pathology and the mechanics of otosclerosis are presented.

The outstanding point made by the author deals with his observations on over 2000 cases and his beliefs relative to failure of fenestration operations in cases which give a good preoperative outlook. He feels that many of these failures may be explained on the basis of involvement of the round window in the otosclerotic process. Diagrammatic illustrations are utilized to explain the mechanical reasons for such failures. This is an excellent presentation, which should be read by all interested in this subject.

LEJEUNE and STEVENSON, New Orleans.

CARCINOMA OF THE MIDDLE EAR AND MEATUS. J. W. S. LINDAHL, J. Laryng. & Otol. **69**:457 (July) 1955.

This discussion is limited to carcinoma of the middle ear and meatus and excludes lesions of other histologic types and lesions of the pinna. It deals with early and late symptoms, assessement of results of the present therapy plans, and a proposal of a possible approach to the problem in the future. The basis upon which the discussion rests is two series of cases, namely, 16 cases known to the author and unpublished previously and 89 cases that the author collected from previously published reports.

Among the important conclusions reached are that pain and bleeding are early symptoms of importance; that early combined surgical and radiation therapy have been the best therapy; that most cases are diagnosed relatively late; that extension to surrounding structures is an important, and often fatal, feature; that perhaps early very radical surgery would offer the best results. He suggests centralization of treatment facilities for this rare lesion.

LEJEUNE and STEVENSON, New Orleans.

DIFFUSE EXTERNAL OTITIS: PATHOGENESIS AND TREATMENT. BEN H. SENTURIA, Laryngoscope **65**:313 (May) 1955.

In order to understand external otitis, the anatomy and physiology of this region must be understood. The kind of skin and glands present is particularly important. Senturia reviews these structures in detail. The physiology of the glands as well as the nature of the secretions is also considered.

Normal subjects in hot humid atmosphere were observed. Very little cerumen was noted, probably because of frequent swimming and the rapid spread of the lipids on the warm, moist skin. Heat and high humidity may cause a change in the pH toward the alkaline side, with resultant hyperkeratosis and plugging of the glands. All these are factors in the etiology of external otitis. Senturia then reviews the histopathology of this condition.

Therapy should include the following: (1) prophylactis in susceptible persons during hot humid weather and (2) topical and/or systemic treatment early. This should be soothing and palliative in the mild stages and active and curative in the severe stages.

Included in the above are removal of the patient from the hot humid weather, cleansing of the ear canal, application of mild astringents and drying solutions, and nonirritating antipruritics combined with antibiotics of low-sensitivity index. In the severer cases, the antibiotics should be used systematically and a wool fat (lanolin) or glycerin pack inserted into the ear canal for 24 hours to reduce the edema. Cleaning treatment should be continued throughout.

HITSCHLER, Philadelphia.

EVALUATION OF TRANSTYMPANIC MOBILIZATION OF STAPES AS A TREATMENT FOR OTOSCLEROSIS. ALAN AUSTIN SCHEER, New York J. Med. **55**:2609 (Sept. 15) 1955.

Scheer works in the Conservation of Hearing Clinic at the Manhattan Eye, Ear and Throat Hospital. This paper was read at the Annual Meeting of the State Medical Society May 13, 1955. His technique is essentially that of Samuel Rosen, who has taught it to a number of otologists and wishes as many as possible to try it out. The author has had 50 cases under observation for one year. Forty-seven stapes were fixed when approached during surgery, of which 31 were successfully mobilized. Seven illustrations are presented to help clarify the text. Group A contained those whose speech frequencies were above 30 db. (15 patients); Group B, those less fortunate (16 patients). Since local anesthesia is used, release of the stapes at operation brings a dramatic expression of relief by the patient at once. Of the 15 cited above, 12 have maintained the original improvement from 12 to 14 months. Two have dropped back, so that their gains are about nil. A drawback is possible fracture of the stapes during attempts at mobilization because of too great fixation. However, such patients may still be offered the fenestration operation, with a good degree of expected improvement. It is noteworthy that 30% of the 50 patients operated on had hearing up to the 30 db. level for speech. In the discussion Dr. Samuel Rosen said that in his 600 operations there have been no complications, especially no labyrinthitis. "The risk to the patient is almost nil." Long-period fixation naturally makes release more difficult, and therefore good results are less certain (20 to 35 years' fixation). A shorter period (3 to 10 years) favors restoration after mobilization, but there are, of course, some exceptions. In general, bone conduction is better than air conduction in otosclerosis; therefore, Rosen does not agree that air and bone conduction tested by the 1024 fork should be about equal. Rosen stresses the importance of exhaustive preoperative tests and a complete audiologic evaluation by speech audiometry. Calibration of equipment, selection and calibration of proper masking for bone-conduction tests, control of test conditions, and skillful testing by an audiologist who is not going to be the operating surgeon offer the best grounds upon which a later evaluation of surgical results can be based.

ABSTRACTOR'S COMMENT: Rosen has often stated that mobilization of the stapes is essentially a "simple operation"; yet it seems difficult when one reads about it. One satisfying thing is that it is not the last recourse: If a patient is fit for stapedial release and cannot get it, there is still the fenestration procedure in prospect. However, the latter is never "simple," according to most operators, and certainly the patients seldom think so. In former days, we were all cautioned against touching the stapes while doing a radical mastoid because of a possible conversion into labyrinthitis. According to Rosen, this is not a warranted fear. Since this stapedial release procedure is quite new, complete evaluation of the results must wait for perhaps another five years or so. It is encouraging to learn that so many of the younger men are working up the technique. There is so much to recommend such an "exploratory procedure."

I. W. VOORHEES, New York.

AUDITORY INVOLVEMENT IN DIVERS AND CAISSON WORKERS. J. COLEMAN SCAL, New York J. Med. **55**:2769 (Oct. 1) 1955.

Decompression in caisson workers sometimes is synchronous with pressure deafness and aural pain. Three types are recognized: (1) transitory middle ear involvement from faulty or too rapid decompression, (2) temporary deafness with or without vertigo (In severe cases there is hemorrhage into the drum and middle ear), and (3) permanent deafness simulating Ménière's syndrome with vertigo, nausea, and vomiting. Air bubbles in the cochlea and vestibule are competent causes of this disaster. Compression causes "gas," present in the middle ear, to expand, causing a bulging drum with pain and sometimes rupture. If the Eustachian tube remains closed, the only escape for gas or air is via a perforated drum. In decompression, the reverse order maintains, that is, diminished hearing, tinnitus, pain, and fullness in the ears. All of these symptoms may be present before the candidate undertakes his underwater occupation; therefore, a thorough ENT study should precede employment. Hearing loss is likely to persist after "the bends" have been apparently nullified. The "bubbles" are from nitrogen or carbon dioxide. When migraine occurs, there is probably vascular spasm of the cerebral blood vessels, with marked loss of bone conduction and of hearing in the upper-tone levels. The hearing loss varies from 25 to 35 db. Five case reports are given to illustrate individual problems. Because of the medicolegal aspects and awards of compensation, it is imperative that the preemployment status of the entire ENT areas be set down.

I. W. VOORHEES, New York.

CONGENITAL DEAFNESS AND HAEMOLYTIC DISEASE OF THE NEWBORN. L. FISCH and D. A. OSBORN, Arch. Dis. Childhood **29**:309 (Aug.) 1954.

In an investigation of 891 children with congenital perceptive deafness, evidence of hemolytic disease of the newborn was found in 27, of whom 20 had varying degrees of choreoathetosis. There appeared to be some correlation between the degree of deafness and the severity of other neurological signs.

These cases were compared with other cases of congenital perceptive deafness associated with anoxia and birth injury (83), prematurity (68), heredity (50), and maternal rubella (69). A similarity was noted between the audiograms of the cases associated with hemolytic disease and anoxia, respectively, in both of which there was selective high-frequency loss. It is suggested that in these two groups there is at least a common site of the lesion if not a common etiology.

KELLY, Milwaukee [A. M. A. AM. J. DIS. CHILD.].

Pharynx

ALLERGY IN ITS RELATION TO THE TONSILS AND ADENOIDS. KENNETH L. CRAFT, Laryngoscope **65**:322 (May) 1955.

Allergy often causes symptoms formerly attributed to diseased and enlarged tonsils and adenoids. Often by recognition and treatment of the allergy, surgery is avoided. A careful history, skin tests, and examination of the nasal mucus for eosinophiles will help in diagnosis Occasionally there is sensitivity to one's own bacteria, indicating the use of an autogenous vaccine.

It is thought too that allergy predisposes to the recurrence of lymphoid tissue in the throat after surgical removal.

HITSCHLER, Philadelphia.

THE TOPICAL USE OF HYDROCORTISONE OINTMENT FOR CERTAIN ORAL CONDITIONS. A. A. FISHER, New York J. Med. **55**:2494 (Sept. 1) 1955.

The author is a dermatologist working in the New York University Post-Graduate Medical School and Hospital. He reports the experiences of various men in treating lesions of the skin, body orifices, eyes, and ears (external otitis). Aqueous and saline suspensions were tried, concentrations varying from 0.25 of 1% to 2.5%. These were put aside in favor of an ointment consisting of hydrocortisone acetate in petrolatum. Treated were less than 50 patients with conditions varying from leucoplakia (active and inactive) through lichen planus, aphthae, denture lesions, glossitis, glossodynia, cheilitis, and disturbances due to toothpaste and lipstick. Patients were advised to rub the ointment vigorously into the lesions three times a day. The drug was found to be acceptable and nonirritating. There was a tendency to heal fissures and a leveling effect on raised patches, but from three weeks to one month was required to gain a satisfactory status.

ABSTRACTOR'S COMMENT: This may be regarded as an experiment with a new modality. Aside from some fair results in "inactive" leucoplakia, there seems to have been little or no good results in any of the other conditions mentioned above. The toothpaste and lipstick conditions recovered as soon as the cause was removed regardless of hydrocortisone. Nothing is said of "trench mouth," which would prove a real test if efficacious. This report has little to recommend hydrocortisone in oral conditions. It is also questionable whether one ought to experiment with such a powerful agent, which may be swallowed in considerable quantity.

I. W. VOORHEES, New York.

THE CLINICAL SIGNIFICANCE OF THE RIDGE OF PASSAVANT. R. T. BARTON, Surg. Gynec. & Obst. **101**:317 (Sept.) 1955.

The author calls attention to a fold in the nasopharynx which has been given little attention by laryngologists, speech therapists, and plastic surgeons. This anatomic structure, known as the ridge of Passavant, was described in 1863 in one of the early treatises on speech physiology. Barton presents his observations from numerous dissections on the nasopharynx and oropharynx and from studies made during surgical procedures in this area.

The ridge of Passavant is described as a transversed fold of the posterior pharyngeal wall which develops during speech, swallowing, retching, vomiting, or whenever it is necessary to shut off the nasal cavities from the oral cavity. The line extends across the entire breadth of

the posterior pharyngeal wall, including the raphe. The exact nature of the fold has been a matter of conjecture, but the author feels that the muscle responsible for this physiological and anatomical structure is almost exclusively the superior constrictor of the pharnyx. He points out that a section of the superior constrictor shows this upper border to be slung somewhat like a hammock from the median pterygoid plate across to the median raphe. The interval between the upper border of the muscle and the base of the skull is closed by the pharyngeal aponeurosis. When the superior constrictor contracts, it is pulled up from its sagging position to a straight line, obliterating much of this. As the muscle pulls upward it tends to carry the overlying mucous membrane with it, buckling it against the base of the skull. The author looks upon this ridge of Passavant as being extremely important in closure of the nasopharnyx. The soft palate and uvula became apposed to the posterior pharyngeal wall and the ridge of Passavant. The ridge appears with the contraction of fibers of the superior constrictor and possibly the palatopharyngeus muscles laterally.

The author has pointed out that nasality after adenotonsillectomy has usually been attributed to damage to the pillars caused during surgery. He feels that it is more likely to occur because of the complete removal of the ridge of Passavant with the adenotome. To substantiate this thesis he points to examples where the entire posterior pillar has been inadvertently removed and yet there was no inability to close the nasopharynx. This was due to the fact that the ridge of Passavant had been left intact and the superior constrictor muscle was able to form it.

In summary, the author calls attention again to the nasopharyngeal sphincter formed by the levators pulling the palate upward and back, by the palatopharyngeus pulling lateral pharyngeal walls inward, and by the ridge of Passavant bulging anteriorly from the posterior pharyngeal wall. Because of its importance in articulation and deglutition, this area should be subjected to a minimum of trauma if postoperative speech and swallowing difficulties are to be avoided.

FRIEDBERG, Chicago.

LATE REPAIR OF CLEFT PALATE BY MAXILLARY OSTEOTOMY, WITH TUBE PEDICLE CLOSURE OF THE NASOBUCCAL FISTULA. P. CLARKSON and W. G. GROSSSMAN, Brit. J. Surg. **42**:206 (Sept.) 1954.

A case is described here of a severe persistent cleft palate and lip in a young male patient. Besides the open cleft in the palate, there was marked malocclusion and severe speech disability. The method of Gillies was followed, with reduction of the collapsed arch by osteotomy and closure of the enlarged cleft in the palate by transporting soft tissue from a distance, as a tube pedicle raised from around the lower surface of the neck, where scars are more easily concealed in men. The procedure is well illustrated by line drawings and photographs.

SELTZER, Philadelphia.

Larynx

MYOBLASTOMA OF THE LARYNX. I. P. J. MACNAUGHTAN and M. S. FRASER, J. Laryng. & Otol. **68**:680 (Oct.) 1954.

An extensive sessile tumour on the posterior wall of the larynx caused hoarseness and stridor in a boy aged 9 years. It was recognized as a granular-cell myoblastoma and is the first example to be reported in the larynx of a child. The growth was excised by laryngofissure but recurred seven months later. Radiotherapy was unsuccessful, and laryngectomy was ultimately required. Analysis of previous reports of laryngeal myoblastoma, with reference to clinical features and treatment, shows that this was an exceptionally severe case.

FROM THE AUTHORS' SUMMARY [A. M. A. AM. J. DIS. CHILD.].

ORGANIC LESIONS OF THE LARYNX PRODUCED BY MIS-USE OF THE VOICE. GEORGE B. FERGUSON, Laryngoscope **65**:327 (May) 1955.

Laryngologists should be aware of and understand better the use and misuse of the voice. Many complicated forces must be integrated to produce the voice, and, likewise, multiple varieties of interference with the voice are possible. The diaphragm, chest, and local muscles all play a part. Tension or misuse of any these would result in interference. Likewise, constriction of the airway will result from improper position of the head (bent forward) during phonation. Certain

types of occupation (those requiring frequent use of the voice) coupled with certain types of personality (high strung, nervous) combine to produce spastic dysphasia, and eventually organic lesions. Such organic lesions are the vocal nodule, contact ulcer, and certain types of diffuse laryngitis. Treatment cannot be confined to local care of the larynx but must embrace voice training and psychic analysis. Only by the inclusion of these latter two will a permanent cure result.

HITSCHLER, Philadelphia.

THE TREATMENT OF CANCER OF THE LARYNX. GLENN H. LEAK, New York J. Med. **55**:2515 (Sept. 1) 1955.

This is the seventh article of The Treatment of Cancer Series put out by the Subcommittee on Cancer of the Medical Society of the State of New York in cooperation with the State Department of Health. The article is based on work done in the various institutions in Buffalo. Leak states that 2% of all human malignancies are in the larynx and that this type of cancer should be curable because of its accessibility. He accepts the terminology of intrinsic and extrinsic, according to whether the visible lesion involves the true cords, the ventricles, or the subglottic regions or whether it extends into the pyriform sinuses, postcricoid area, or, presumably, into the gland of the neck. The author thinks that "many cases . . . can be cured by irradiation, if well selected and treated by a well qualified radiotherpist. . . ." He believes that many patients with early cancer of the larynx can be cured by irradiation. For extrinsic cases, he recommends surgery, laryngectomy, with subsequent irradiation as required by the subsequent progress. Contraindications are cardiac failure, and great debility. When surgery is refused utterly by the patient, irradiation is the only recourse. But one must remember that a chondritis may intervene under massive dosage. Laryngofissure seems to have a high recurrence rate, although it may be used when only one cord is involved near the anterior commissure but not crossing this area, and without fixation. Total extirpation should be done in posterior commissure lesions with infiltration of arytenoids and fixation. Then one should do block dissection of the cervical glands and lymphatics. Recently this radical surgery has improved because of the intratracheal tube, whole blood transfusions, antibiotics, and better preparation of the patient, with conscientious nursing and observation after operation. Five-year survivals are not uncommon of late, whereas a three-year hold-out was formerly regarded as highly successful. Clerf, of Philadelphia, is quoted in this regard as having five-year survivals in 34 cases (65%). One must remember that cancer may be primary in the upper esophagus and unseen, extending into the larynx surrepititiously, so that it may seem to be primary in the larynx instead of secondary. These are the delusive instances. Recurrences do try the skill and patience of every surgeon, but there is little hope in doing further surgery unless it may be quite minor, for granulations, etc. We are limited to irradiation, massive in type, when death is in prospect. Insertion of gold-tube radon seeds may be of much service. Rehabilitation must be promised to every patient who rejects operation because of prospective loss of voice. It is well to have him see and hear a laryngectomized patient, perhaps before operation but certainly after it. Many hospitals have classes for these victims, some of them taught by teachers who have lost their larynges (National Hospital for Speech Disorders, Postlaryngectomy Division). In any case of hoarseness, mirror study must be completely carried out. Barring that, direct tube examination is obligatory.

ABSTRACTOR'S COMMENT: There seems to be slight confusion in the author's mind when he says ". . . the overall results of the treatment of laryngeal cancer by irradiation therapy are disappointing." Then he proceeds to recommend it in highly selected cases. But he admits, as we all must, that there is no sharp dividing line between intrinsic and extrinsic cancer. Not until we get a complete view of the interior of the larynx, as is afforded by laryngofissure, can we be quite sure of the extent of the lesion.

I. W. VOORHEES, New York.

TRACHEO-ESOPHAGEAL FISTULA WITHOUT ATRESIA. GEORGE WILLIAM WARE and LOUIS L. CROSS, Pediatrics **14**:254 (Sept.) 1954.

Congenital tracheoesophageal fistula without atresia of the esophagus is a rare anomaly. Twenty-six patients are reported in the literature. The authors report an additional case. The etiology is still obscure, but may be due to the development of aberrant blood vessels.

Patients present a triad of signs and symptoms, coughing and cyanosis with feedings, abnormal distention of gastrointestinal tract, and persistent pneumonitis.

Radiographic investigation is most important. Iodized oil (Lipiodol) study of the esophagus in the oblique or prone position is the most rewarding diagnostic test.

Surgery is the only method of cure.

FROM THE AUTHORS' SUMMARY [A. M. A. AM. J. DIS. CHILD.].

NON-INFLAMMATORY LARYNGEAL STRIDOR IN INFANTS. JAMES CROOKS, Arch. Dis. Childhood **29**:12 (Feb.) 1954.

"Congenital laryngeal stridor" is a clinical description and should not be used to denote the specific entity of inspiratory laryngeal collapse of infants. This condition is described and also another developmental abnormality the baby outgrows, which the writer calls inspiratory arytenoid prolapse.

Case records are given of eight babies with laryngeal stridor due to localized conditions in the larynx, open to surgical treatment and if not so treated likely to prove fatal. Four of the babies had cysts, two hemangiomas, one a cleft larynx, and one a foreign body.

The larynx of a baby with stridor should be inspected to establish the cause.

The difficulties and dangers of tracheostomy in young infants are discussed.

KELLY, Milwaukee [A. M. A. AM. J. DIS. CHILD.].

Nose

NASAL GLIOMAS. F. E. ALTANY and K. L. PICKRELL, A. M. A. Arch. Surg. **71**:275 (Aug.) 1955.

The authors present a case of a 7-week-old white male infant with a firm, elastic, circumscribed tumor at one side of the nasal bridge. The tumor was present at birth, which was a normal labor. An operation was performed under orotracheal anesthesia, through a transverse incision across the bridge of the nose. A firm, homogeneous, light-yellow mass was removed entirely, with a wide margin of normal skin. Frozen sections indicated glioma. The incision was closed with fine white and black nonabsorbable (silk) sutures. There were no complications, and the patient was discharged on the second postoperative day. There are excellent photographs showing the condition before and after operation. These tumors are considered to be encephaloceles which become isolated during fetal brain development.

SELTZER, Philadelphia.

A NEW MATERIAL FOR IMPLANTATION IN CASES OF ATROPHIC RHINITIS. A. P. SELTZER, J. Internat. Coll. Surgeons **23**:113 (Jan.) 1955.

In atrophic rhinitis, if the process is sufficiently severe, the nasal cavity becomes enlarged to such an extent that an abnormal condition exists. To correct this state of roomy nose, Seltzer devised a method of subcutaneous implantation of Ivalon, which is a polyvinyl sponge. Under local anesthesia, the nasal lining over the turbinates is elevated to form a suitable-sized pocket, into which a shaped piece of sponge is inserted. If there is also atrophy of the septum, the cartilage is split and a small wedge of sponge is placed within it. In this way the normal size of the nasal passage is restored, and the improvement is recognized at once by the patient.

AUTHOR'S SUMMARY.

PLASTIC REPAIR OF THE FRACTURED NASAL SEPTUM. I. MUSKAT, J. Internat. Coll. Surgeons **23**:117 (Jan.) 1955.

In correcting the deformity due to fractures of the nasal septum, Muskat follows in general the procedure described by Seltzer and by Steffenson, a three-step operation, to secure the desired result. The details of the technique will depend in each case upon the individual condition. This involves not only the septum but also the lateral and alar cartilages. These details are given for the different types of cases treated and are illustrated by 35 drawings.

SELTZER, Philadelphia.

THE SURGICAL TREATMENT OF CANCER OF THE PARANASAL SINUSES. EDGAR I. FRAZELL, Laryngoscope **65**:557 (July) 1955.

In the past, surgery for cancer of the paranasal sinuses has been in disfavor because of its disfiguring effect and the difficulty in eradicating the lesion completely. Likewise, radiation has been largely unsatisfactory. Surgery with post operative irradiation recently, however, has a greater chance of success, partly because of the modern improvement in anesthesia, blood replacement, skin grafting, antibiotics, etc.

Cancer of the sinuses rarely involves but one set of air cells. The nasal cavity and maxillary antrum are frequent sites of origin, the ethmoids occasionally, and the frontal or sphenoids rarely. Unfortunately, the signs and symptoms of cancer are rarely evident early in the disease and early radiologic examination is often of little aid.

Most of the cancers are squamous or epidermoid. They may be of all grades. Sarcomas and melanomas also occasionally are found.

Cancer of the maxillary antrum is best removed by extirpation of the maxilla and curettage of the adjacent areas. Whether or not to remove the eye is sometimes a difficult decision. The base of the skull and cribriform plate area may be removed, if necessary, without undue fear of meningitis, because of the antibiotics. Skin grafting with split-thickness grafts following the operation is imperative.

The prognosis of these conditions is difficult to determine and is still poor. Figures vary from 15%-30%.

HITSCHLER, Philadelphia.

THE MANAGEMENT OF CANCER OF THE MAXILLARY ANTRUM. D. RAINES and A. G. JAMES, Surg., Gynec. & Obst. **101**:395 (Oct.) 1955.

The authors outline the management of cancer of the maxillary antrum as carried out on 16 patients with primary carcinoma of the antrum seen from 1949 to 1953. Their surgical treatment is maxillectomy as practiced in most centers at this time. The postoperative therapy consists of a plastic mold containing radioactive substances to the surgical defect for the provision of necessary irradiation.

FRIEDBERG, Chicago.

A LATERAL APPROACH TO THE FRONTAL AIR-SINUS. G. F. ROWBOTHAM, P. R. R. CLARKE, and D. P. HAMMERSLEY, Brit. J. Surg. **42**:524 (March) 1955.

The authors have found the operation described by them to be useful in chronic and acute infections of the frontal sinus and suggest that it may be successful also in the treatment of chronic catarrh. The technique is illustrated by the presentation of a patient, a young man of 39, who had a hard mass about ¾ in. in diameter projecting downward from the upper left orbital margin, with downward displacement of the left eyeball. The fundus was normal. The diagnosis of a large hard neoplasm was made, and an operation was carried out through an S-shaped incision extending upward from the left superior orbital ridge through the hairline, then through the attachment of the temporal fascia. Raising the muscle flap exposed the external angular process, the malar bone, and part of the temporal fossa. A burr hole was made through the bone over the anterior fossa, and the tumor mass was removed through a further opening. Drawings show the detail of the operation, which resulted successfully, with only very slight residual deformity.

SELTZER, Philadelphia.

Miscellaneous

MANAGEMENT OF CANCER OF THE HEAD AND NECK. J. J. LONGACRE, J. LEICHTLER, and P. JOLLY, J. Internat. Coll. Surgeons **23**:1 (Jan.) 1955.

This is a significant paper based on the study and surgical treatment of 107 cases of head and neck cancer. Each one is a study in itself, and, consequently, no detailed review is attempted. Recurrence was present in only 4.6% of the cases. Various photographs show the original condition, the methods used, and the postoperative results. An interesting point is made in the description of the means used to raise and sustain the morale of each patient: showing them the plan proposed for reconstruction and the results to be anticipated therefrom.

SELTZER, Philadelphia.

MANAGEMENT OF FACIAL SCARS. J. R. R. LEWIS, J. Internat. Coll. Surgeons **23**:78 (Jan.) 1955.

Lewis presents a group of different types of facial scars due to various causes, which are repaired by an operative technique suited to the individual case. There is a wide variation in their nature and degree. In the greater number more than one operation was required, all of which were done under local anesthesia. In the severer conditions surgery was required; but in others of less involvement treatment was given by abrasion, the latter particularly for the removal of smaller scars. The authors observed that the improvement of the appearance of the skin of the face usually led to a desirable change in the personality of the patient, who acted favorably in both social and work relationships. Sixty-eight photographs illustrate the work presented.

SELTZER, Philadelphia.

STRESS AND DISEASE. HANS SELYE, Laryngoscope **65**:500 (July) 1955.

For years it has been known that stress and disease are related. Recent experiments have demonstrated that the organism responds in a stereotypical manner to a variety of widely different factors, such as infections, intoxications, trauma, nervous strain, etc. All these factors have a common feature: They put the body in a state of general stress. The stereotypical response represents the somatic manifestations of nonspecific "stress" itself. The chief manifestation of this stress response is an involutional or degenerative change in all the organs save the adrenal cortex, which shows hyperactivity. The above reaction, however, is but the beginning of a general adaptation syndrome. There is a stage of resistance and a stage of exhaustion. The whole process is dependent upon the hypophysis adrenocortical system, which may either enhance or inhibit the body's defense reactions against stress. Derangements of this system produce the so-called diseases of adaptation.

HITSCHLER, Philadelphia.

THE PROPHYLAXIS OF ALLERGIC DISEASE WITH SPECIAL REFERENCE TO THE NEWBORN INFANT. JEROME GLASER, New York J. Med. **55**:2599 (Sept. 15) 1955.

Glaser says that most allergic diseases begin in childhood and ascribes this to the diet of the pregnant woman. He asks his pregnant mothers to avoid eggs and custards, to drink a pint of boiled milk each day, and to eat no cheese. A variety of meats is allowed for protein content, and he adds calcium and phosphorus to their diets as necessary. Lack of breast feeding (and the substitution of cow's milk, therefore) is contributory to skin conditions (eczema) in infants. Egg yolk is not allowed the infant until the 6th or 9th month, during which period some immunity against egg yolk may be expected. The same is true for egg white. Many items ingested by the nursing mother are present in the mother's milk. Therefore, the parental diet during pregnancy should be maintained during the first postnatal months. There is no condemnation of cow's milk, for it is a "great gift to mankind," but in certain infants it is unwelcome and may cause various troubles in the digestive tract or skin. However, the gradual abandonment of the female breast as Nature's food emporium for infants may possibly be responsible for some evils now overlooked as to etiology.

ABSTRACTOR'S COMMENT: ENT specialists doing allergy must often be puzzled as to management of allergies in very young patients. Since anything which gets into the mother's blood stream may affect the infant for good or ill, what about tobacco and alcohol, so commonly in use these days by women as well as by men?

I. W. VOORHEES, New York.

RADICAL SURGERY IN ADVANCED CANCER OF THE LUNG. ADRIAN LAMBERT, New York J. Med. **55**:2606 (Sept. 15) 1955.

Lambert works at Roosevelt and Bellevue Hospitals and also at the College of Physicians and Surgeons of Columbia University. In 14,000 consecutive autopsies at Bellevue, 16% showed cancer, and of this number 23% revealed primary cancer of the lungs. In males, 1200 of whom were studied postmortem, some 30% had primary carcinomas of the lungs. While cancer of the stomach used to be the number one cancer killer, it is now in second place, with an incidence of about 15%. Analytically, one male in three who dies of cancer has it in his lungs. Since the GP sees the "chest case" first, it is up to him to make an early diagnosis, but there are many reasons for overlooking an incipient lesion. The usual means of physical diagnosis (palpation, auscultation, etc.) are, after all, rather crude. We must depend upon bronchoscopy, biosection, and x-ray. These, too, may not be determinative, but we are sure that if metastasis has already

occurred the possibility of "cure" becomes more remote by whatever method of therapy is employed. Cough, hemorrhage, and pain may come on late in the disease, and all of these are likely to be misinterpreted. In x-ray film studies, any thickening of the hilar areas should make them suspect of malignancy. But these lesions may resemble unresolved pneumonia, emphysema, or tuberculosis. Bronchoscopy with biopsy when possible, and the Papanicolaou smears may be of service. Surgery is the best method of management, but it must be done early. There are some contraindications: Any tumor not localized in the thorax and which is not primary will not benefit by surgery. Pulmonary insufficiency will be worsened by removal of lung tissue needed for breathing; hence, the patient may become a "pulmonary cripple." In the presence of an overwhelming disease, such as tuberculosis, cancer of the lung is of secondary importance and is inoperable. There are four surgical procedures which can be offered to the patient: Where the cancer is confined to one lobe, lobectomy may be first choice. Pneumonectomy may be performed when the cancer is chiefly at the hilum. Radical lobectomy with removal of all possible lymph glands and lymphatics will include also right and left chains of bronchial lymph nodes, carinal nodes, and the paratracheal chains. This radical procedure is the method of choice provided the patient can tolerate that much surgery. A fourth surgical approach is palliative resection in the presence of pulmonary suppuration and excessive hemorrhage. The patient will have symptomatic relief for a time, although he will die later of metastases. During the past five years, this radical operation has been so greatly improved technically that one may speak of "cure." Surgeons have learned from experience just how to manage the mediastinum at operation.

In considering the operability of a cancer, there are three groups into which the patients fall: In the first group, where cancer has extended beyond lung tissue and into the carinal, paratracheal, and bronchial nodes without involvement of adjacent organs, surgery offers much. In the second group there is extension into adjacent organs which can be resected, with a five-year survival expectation. In the third group the cancer extends to vital organs, such as the vena cava, esophagus, and trachea, which, if removed, require some kind of prosthesis or substitute. This group represents the most desperate extension of the disease, invasive of the chest wall, and the above-mentioned vital structures. The surgical risk is great, without commensurate results.

Radiation is valuable where surgery is not acceptable, but no cancer of the lung has been cured by radiation alone, according to Lambert, while cases with mediastinal extension who have been operated on have been cured by surgery. High-voltage therapy has given promise but should not be instituted until six weeks have elapsed following surgery. To summarize, at this time "surgery is the only means of cure for this disease." Moreover, the cure is in ratio to the stage of the disease at time of operation. Finally, good results have been obtained by surgery where the process has extended beyound the lung into the chest wall and into the mediastinum.

ABSTRACTOR'S COMMENT: This is an incisive, direct, coherent report on surgery of the chest for cancer of the lung, with rather more sense of assurance as to the ultimate benefits than can be found in most studies of this kind. The word "cure" is used more often than one commonly finds in discussions on cancer.

I. W. VOORHEES, New York.

TREATMENT OF PRIMARY CANCER OF THE LUNG. WALTER F. BUGDEN and J. ERNEST DELMONICO JR., New York J. Med. **55**:2645 (Sept. 15) 1955.

This is the eighth in a series on the Treatment of Cancer, under the aegis of New York State authorities on Public Health.

Primary bronchogenic cancer is rapidly increasing, not only statistically but actually and really. Primary lung cancer comprises 10% of all malignancies and is the commonest form of visceral cancer in males; 20 times more frequent than in females! The recent cigarette scare, based as it was on carefully prepared statistics by the highest authorities, is waning, because it is impossible to state absolutely that smoking is the chief cause of cancer of the lung. Proof now adduced is sufficient to convince physicians of the dangers of smoking but not sufficient to frighten the public, which wishes to keep on smoking, regardless. A simple working classification from pathology is epidermoid or squamous, anaplastic or undifferentiated "oat-cell" cancer, adenocarcinoma. Grossly the tumor may appear as a localized mass, an abscess cavity, an invasive mass, a pneumonic infiltration, etc. "There is no typical picture."

We should like to make an early diagnosis in the "silent phase," but there are neither symptoms nor signs which are specific. Cough, pain, wheeze, and hemorrhage are all late in coming. X-ray of everybody's chest is fairly impossible but might be required of all smokers of 40 years

and over every six months or oftener. Nonsmokers must still take the usual chance of dying from cancer or something else. Thoracic surgeons take the seemingly extreme view that all suspicious-looking x-ray films of the lungs better be questioned for cancer. Nothing much is said by them about tuberculosis. "Round, silent x-ray shadows in all individuals over forty are often malignant (from 20 to 50 percent)." Surgeon Graham of St. Louis performed the first successful pneumonectomy in 1933 on a physician who is said to be still living. Diagnosis? The mortality after some 23 years is only about 10%-15% today. Reasons: better positive pressure anesthesia, blood replacement, better surgical techniques, and antibiotics given before and after surgery to reduce secondary infection.

Bugden and Delmonico claim that 50% of the patients they see are inoperable because of distant hematogenous or lymphatic metastases, hoarseness which shows invasion of the recurrent laryngeal nerve (left thorax), reduced cardiorespiratory function insufficient for survival, bronchoscopic evidence of extension of cancer into the carina or trachea, bloody pleural effusion, paralyzed diaphragm. "So we are able to resect only about 25 percent of all cases seen." Even so, the results are palliative, not curative. The surgeon will not know how much to excise until he gets into the chest and analyzes the conditions found. The family may clamor for "something to be done" and vitiate the surgeon's judgment.

The point is made that sometimes one may use nitrogen mustard intravenously. A subsequent sentence declares that this measure is "very disappointing." As for radiology, not too much can be expected even from massive doses, but "in some cases there is a salutary effect."

Until we have blood studies or radioisotopes or some other means for early diagnosis, cancer of the lung remains still undefeated in its capacity for destroying human life.

I. W. Voorhees, New York.

Recent Advances in Physiology of the Esophagus. Marvin H. Sleisenger, Murray Davidson, James H. Pert, and Thomas P. Almy, New York J. Med. **55**:2747 (Oct. 1) 1955.

This research was carried out in the Departments of Medicine and Pediatrics, New York Hospital-Cornell Medical Center and was reported before the Annual Meeting of the Medical Society of the State of New York at Buffalo on May 12, 1955. The physiology of swallowing and the propagation of peristaltic waves down the esophagus are discussed. Cardiospasm is regarded as a neuromuscular disorder in the esophagus in which there is defective parasympathetic innervation. In differentiating this lesion from cancer, Mecholyl seems to be of value. Esophageal motility is influenced by infiltration of the submucosa and subsequent muscular atrophy. While dysphagia is an important symptom, it may be caused by the so-called "contraction ring" of the distal esophagus, "the exact nature of which is as yet obscure." Eight figures and some 16 references are set down in explanation of the problem at hand. This is of interest to the esophagoscopist whose visual findings may be completely negative as to the causation of cardiospasm in a physical sense.

I. W. Voorhees, New York.

Amebic Lung Abscess. Charles A. Ribaudo, New York J. Med. **55**:2804 (Oct. 1) 1955.

Ribaudo reports one case of amebic lung abscess from St. Vincent's Hospital, New York. The patient was a Chinaman, a restaurant waiter, seen in 1949 with acute pain in the chest, hemoptysis, and cough, but of a duration of only one day! A thorough work-up yielded surprisingly few positive signs, and, although a working diagnosis of hematogenous amebic lung abscess was suggested, the actual parasites were not found until three months after the initial studies were made. Antibiotics offered fairly negative results as to relief. Bronchoscopy was about negative save for "an inflammatory area below the orifice of the right upper lobe bronchus." After all other remedies had failed, it was decided to try emetine, just to see what might happen (the causative trophozoites had not yet been found). The response was dramatic. Pain, cough, and bloody sputum almost ceased, appetite improved, and an increase in weight was found. There were small multiple lung abscesses seen by x-ray, which eventually cleared. There seems to have been an amebic invasion of the liver, with extension to the right lower lobe of the lung. Chloroquine was ineffective, but emetine, administered for 12 days, brought an amazing relief. The author concludes: "In cases of pulmonary abscess in which the usual causative agents have been excluded and which do not respond to penicillin, sulfonamides, or streptomycin, a high index of suspicion for amebiasis must be entertained."

I. W. Voorhees, New York.

CAPILLARY HEMANGIOMA OF THE SKIN. J. J. MODLIN, Surgery **38**:169 (July) 1955.

The author reports a series of 52 consecutive cases of congenital hemangioma of the skin. He considers that such vascular lesions should be completely removed by surgery, without delay or treatment by irradiation, if they are seen at birth or before rapid growth has taken place. If they are seen only after such growth has occurred, the value of the surgery is questionable, since they will regress spontaneously in time, as seen by him in children from 1 to 4 years of age. No such condition was seen after 7 year of age, and this strawberry type of nevus is said never to have been observed in an adult. A complete tabular record of the cases is given, and six photographs, each in color and in black and white, illustrate the paper.

SELTZER, Philadelphia.

AN ANALYSIS OF TWO HUNDRED FIFTEEN CASES OF BRONCHIECTASIS. G. E. LINDSKOG and D. S. HUBBELL, Surg., Gynec. & Obst. **100**:643 (June) 1955.

The authors present data from a series of 215 consecutive cases of proved bronchiectasis, with particular reference to age at onset of the disease and at time of treatment, sex, race, recognized etiologic factors, chief symptoms, anatomic distribution of the disease by lobes and segments, and bacteriology of the sputum. The criteria for selection of patients for surgery are given. Of 165 pulmonary resections of various types carried out on 138 patients, there were 4 hospital deaths, an operative mortality of 2.4%. Follow-up data as well as factors influencing the prognosis of bronchiectasis are discussed. It is interesting to note that of the 214 patients, sinusitis was demonstrated in 74. The cases associated with sinusitis seemed to have an equally good prognosis after resection, whether or not special therapy was directed against the sinus disease before or after surgery.

FRIEDBERG, Chicago.

THE CASE AGAINST SEGMENTAL RESECTION FOR ESOPHAGEAL CARCINOMA. E. F. SCANLON, D. R. MORTON, J. M. WALKER, and W. L. WATSON, Surg., Gynec. & Obst. **101**:290 (Sept.) 1955.

The authors present a very careful analysis of a group of 79 patients who had undergone resection for carcinoma of the esophagus. Study was made to determine the incidence of inadequate resection as based on microscopic evidence of carcinoma at the margin of resection or the development of recurrent tumors in the line of anastomosis.

Of the entire series, 45.6% of the patients were considered to have had an inadequate resection of the esophagus based on the criteria outlined. Of the determinate group of patients in this series (those upon whom all criteria were satisfied) 80% of the patients were considered to have had inadequate resection. It is advocated that transverse rather than longitudinal sections of the margins of esophageal resection should be subjected to microscopic examination routinely. In order to improve the cure rate of carcinoma of the esophagus by surgical methods, the authors advise excision of a greater portion of the esophagus than is now done with routine segmental resection.

FRIEDBERG, Chicago.

BRONCHOGRAPHY WITH DIONOSIL. G. H. C. JOYNT and L. R. HARNICK, Surg., Gynec. & Obst. **101**:425 (Oct.) 1955.

The authors point out that bronchography is a procedure that is generally considered to entail little risk. However, many efforts are constantly being made to improve the media which are used, in order to avoid some of the tissue reactions which may and do occur.

Dionosil is a 50% suspension of an ester closely related to Diodrast. It is available in an aqueous form and in an oily form. Both varieties of Dionosil have the advantage of being rapidly eliminated from the lungs, so that subsequent roentgenograms are not distorted, as formerly occurred in the case of the Lipiodol bronchograms.

Employing Dionosil and using healthy mongrel dogs for bronchography, the authors subsequently killed the animals and studied the lungs microscopically. From their experimental data the aqueous suspension, although somewhat more irritating than the oily, produced less reaction in the lung. It has been suggested that lung resections need not be delayed following a bronchogram with Dionosil, but the results in dogs suggest that three to four weeks should elapse before segmental or lobar resection in the lung is carried out. From their experimental observations it would appear that patchy atelectasis and granulomatous reaction occurred more

consistently with oily Dionosil bronchograms, and there was also a greater tendency for alveolar filling. However, the advantage of its being somewhat less irritating to the mucous membrane should be considered. Good topical anesthesia often avoids any discomfort from the aqueous Dionosil. At the end of four weeks no granulomatous reaction was noted in the lungs of dogs with the aqueous suspension. The aqueous suspension appears to be well tolerated in adults and is nontoxic, although there may be slight febrile reaction lasting up to 48 hours. It may be used in active tuberculosis, without danger, and, finally, patients sensitive to iodine or iodized oils do not react to Dionosil, since no free iodine or iodides are liberated. FRIEDBERG, Chicago.

PARTIAL SECTION OF PROXIMAL SEVENTH NERVE TRUNK FOR FACIAL SPASM. W. B. SCOVILLE, Surg., Gynec. & Obst. **101**:495 (Oct.) 1955.

The author has worked out a technique involving partial sectioning of the seventh nerve in its main trunk just distal to the stylohyoid foramen. The operation has been performed on six patients with unilateral facial tic, and the results have been excellent in one patient, good in three, fair in one, and poor in one. A temporary paresis of the facial muscles for three months and a late mild return of eyelid tic under fatigue for two to four years are to be expected. There have been no complications or permanent weakness. In general the late results have exceeded the expectations of the author. FRIEDBERG, Chicago.

GENERAL PRINCIPLES IN THE USE OF ANTIBIOTICS. WILLIAM J. MARTIN, DONALD R. NICHOLS, and JOSEPH E. GERACI, Arch. med. Cuba **6**:1 (Jan.-March) 1955.

The authors discuss the various antibiotics that are in common use, presenting the various indications and limitations of each. They also stress the problem of combined antibiotic treatment and state that usually the indications are extremely limited. In their experience they believe that a combined treatment may be used for (1) brucellosis, (2) bacterial endocarditis, (3) bacteremia, (4) peritonitis, and (5) meningitis.

Finally, they make a detailed plea against the abuse of these antibiotics. They indicate that the antibiotics have been too often employed for many common ailments, causing a great deal of sensitization and a bacterial resistance, so that when there is a need for them in critical diseases there is an extreme danger due to anaphylaxis and other evidences of side-reactions.

PERSKY, Philadelphia.

NEW IDEAS IN THE TREATMENT OF CAUSTIC INJURIES TO THE ESOPHAGUS. PEDRO HERNANDEZ GONZALO, Rev. cubana oto-rino-laring. **4**:63 (April) 1955.

In discussing the problem of therapy for caustic burns of the esophagus, the author divides the indications for treatment under the following headings: (1) medical treatment and (2) surgical treatment. This is usually a very flexible classification, since the indications for treatment depend primarily on how soon after the ingestion of the caustic the patient is seen. In consequence, the author believes that if the patient is seen within three days following the ingestion of the corrosive medical treatment is indicated. This includes the use of magnesia, milk, albumin water, calcium hydroxide, and olive oil where the corrosive is an acid. If the corrosive is an alkali, he recommends lemon or orange juice, equal parts of vinegar and water, and olive oil. In addition, he claims considerable success with ACTH or cortisone. However, in cases of longer duration and particularly where the burns are more extensive, then endoscopic treatment is indicated. This may be limited purely to drainage, bouginage alone or with either mercury or retrograde dilatation. In a few cases the author found it necessary to do actual surgery. PERSKY, Philadelphia.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. S. Gorrell, 110 Medical Arts Building, Calgary, Alta.

Secretary: Dr. G. Arnold Henry, 328 Medical Arts Building, Toronto.

Place: Chateau Frontenac, Quebec, Canada. Time: June 6-7, 1956.

International Bronchoesophagological Society

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.

Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

Meeting: Fourth International Congress of Bronchoesophagology.

President of Congress: Dr. Antonio Carrascosa.

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Place: Leyden, Netherlands.

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

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Meeting: Fifth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.

Place: San Juan, Puerto Rico. Time: April 8-12, 1956.

President of Congress: Dr. Juan H. Font, Medical Arts Building, San Juan.

General Secretary: Dr. C. E. Muñoz MacCormick, P. O. Box 9111, Santurce 29, Puerto Rico.

Sixth International Congress of Otolaryngology.

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.

General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.

Place: Hotel Statler, Washington, D. C. Time: May 5-10, 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

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Place: Chicago. Time: June 11-15, 1956.

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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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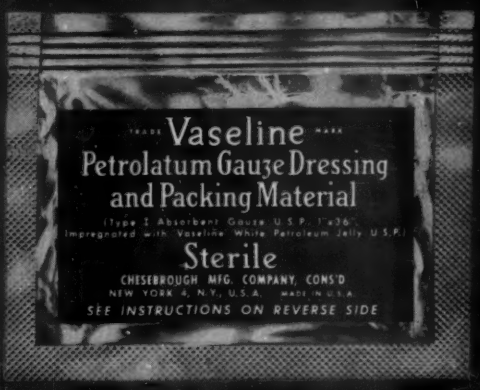
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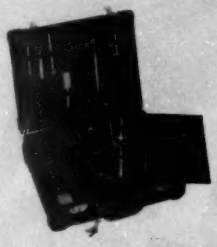

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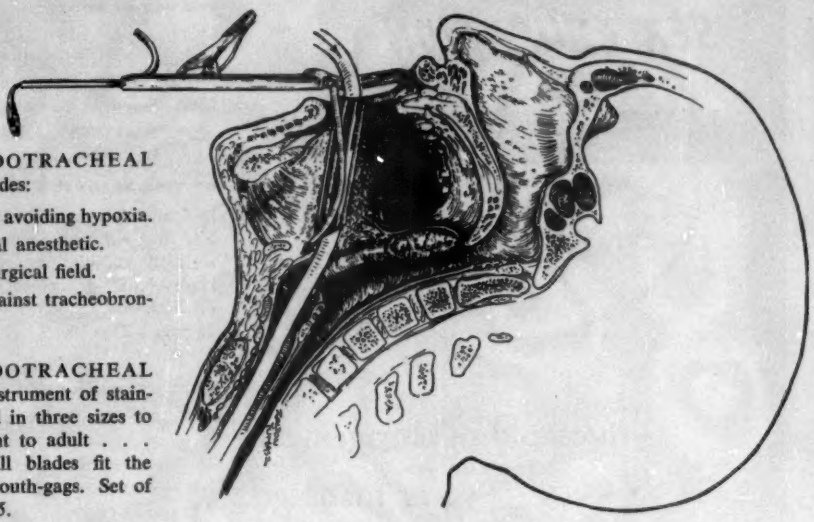

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* Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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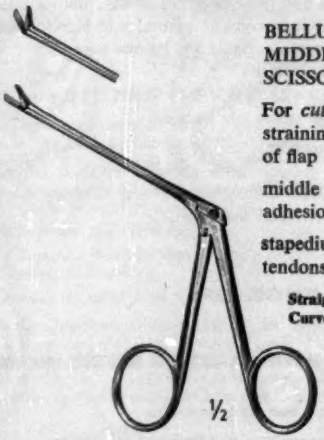

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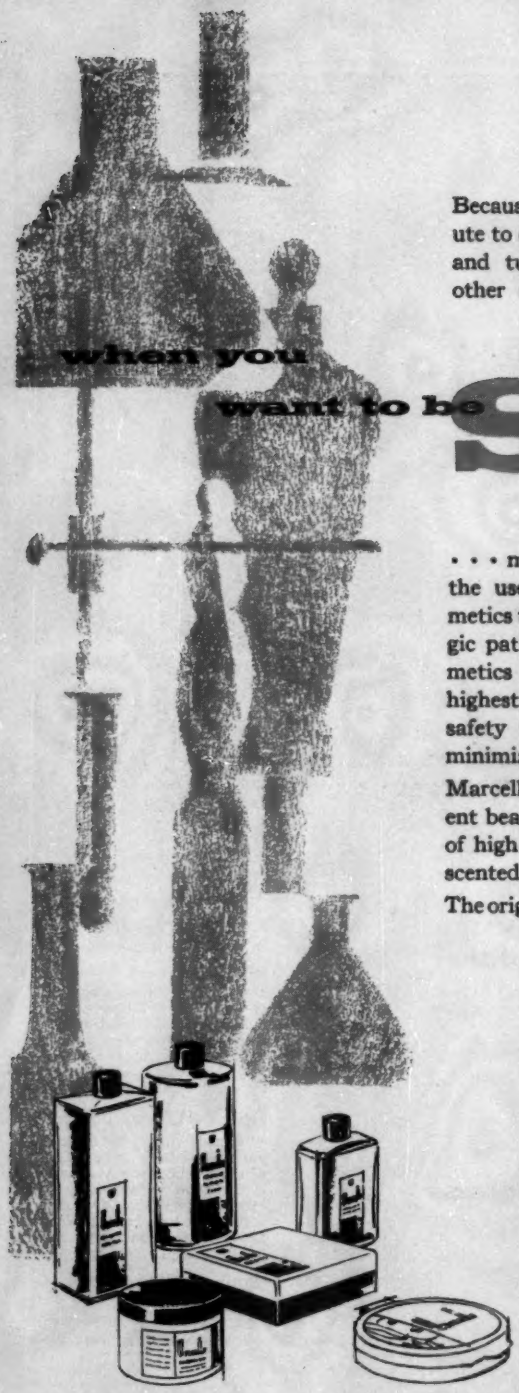

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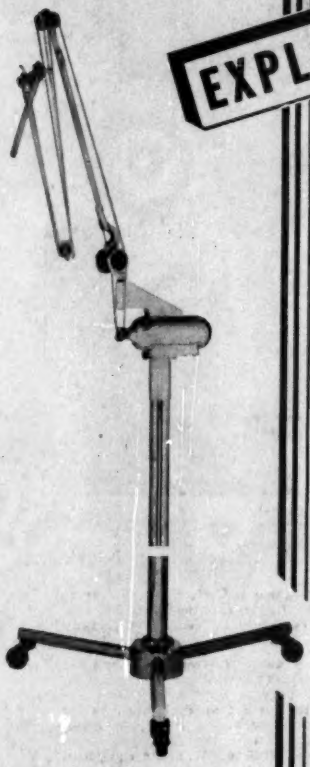

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